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Fungus Diseases of Turf Grasses

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The Cover **— a view of a portion of the turf research plots of the Rhode Island Station. Here factual information for pest control, culture, and varieties is obtained.**

PROGRESS IN TURF DISEASE RESEARCH

KEEPING TURF HEALTHY is a major chore and to think that grass just grows is no longer sufficient. We are becoming more critical and we must have the "know how" to meet the specifications demanded. Certain things stand out in importance; not the least is the judgment and alertness of the man in charge.

Turf culture studies were begun in 1890 at the Rhode Island Agricultural Experiment Station and experimental grass plots have been maintained continuously since then. Damage from disease and insects has become an increasing problem as our knowledge of the conditions favoring the growth of grasses has accumulated. As with grasses, the fungi that injure turf are conditioned by many factors. Cultural practices, site and soil play an important role in the outbreak of disease. Monteith and Dahl (78) discussed this subject so well that it will not be repeated here. With few exceptions, vigorous turf is less likely to become permanently injured by disease than poorly managed turf. However, the repeated injury of mowing cultivated turf offers an ideal infection court for many molds.

RESISTANT VARIETIES

From a growth and aesthetic viewpoint the selection of a superior species, variety, or clone of grass is good practice. However, the exclusive use of a grass, because it is "resistant" to a hitherto serious pathogen, may impart a false, temporary security. Too often other diseases subsequently become even more destructive. For example, we have replaced Chewings fescue, which is very susceptible to anthracnose, with the resistant creeping red fescue only to find the latter killed by pink patch. Seaside bent was substituted for the dollar spot-susceptible Congressional creeping bent (C-19), only to have the Seaside severely damaged by the snow scald fungus. Velvet bent is considered by many as ideal for golf and bowling greens and dollar spot rarely damages it. Yet, copper spot can make it look sick within a few days, and the seed may be up to 80 percent smutted with resultant poor stands and stunted plants. The most practical solution is the planting of a mixture of two or more grasses which are not susceptible to the same diseases. Injury to one is compensated for by the green leaves of the other.

The use of mixed populations of species or varieties to minimize the concentration of disease-susceptible individuals is a principle worthy of emphasis. Farmers and scentists have shown it to be sound practice to use mixed rather than pure stands of grasses for pastures. Although in a mixture of Kentucky bluegrass, fescue, and creeping bent the individual grasses may be susceptible to *Helminthosporium* leaf spot, pink patch, and dollar spot respectively, more than a negligible disfigurement is not likely to occur at any time due to the masking effect of the other members of the mixture.

WEATHER AND DISEASE OUTBREAKS

Experienced greenkeepers are familiar with the sudden appearance of diseases associated with cool or warm, bright or muggy weather. To those whose major interest is growing grass it is clear that precise knowledge of the climate at the soil surface level will permit prediction of the development of some diseases. The installation of air and soil temperature- and moisture-recording instruments and the correlation of disease outbreaks with the data obtained is aiding in better timing of fungicide applications—timely prevention rather than general insurance. The personal skill of the lawnsman cannot, however, be replaced by a printed chart. He must evaluate the effect of air movement, site and cultural practices upon the retention on the grass of the moisture necessary for fungus growth. Some diseases strike when sunlight and resultant carbohydrate reserves in the plants are low, which bespeaks maintenance in top vigor.

FUNGICIDES

The grasses that make up lawns, fairways and greens are attacked by 100 or more diseases. Just as it is unreasonable to expect any one grass to be immune to all of them, so is it to expect one fungicide to control them all. Mercurials were the only effective chemicals available a few years ago for the control of turf diseases. Mercury chloride mixtures (i.e., calomel and corrosive sublimate) are still used most and are very effective when properly applied for brown patch control. Thiuram has been reported to control dollar spot, but in our trials it has not prevented copper spot or dollar spot. Research on turf fungicides has been a major project at the Rhode Island Station in recent years and improved products, including the organic cadmium- and phenyl mercury-containing materials, have been developed. These cadmium fungicides will both prevent and cure cases of copper spot and dollar spot, but they are not effective against brown patch. Heretofore *Helminthosporium* blights have gone uncontrolled, but indications are that the same water-soluble phenyl mercury compounds that are specific for crabgrass will also prevent some of these diseases.

Responsibility has been placed on the manufacturer by the Federal Insecticide, Fungicide and Rodenticide Act for printing on the label the names of diseases which the product will control. Determining how well they perform this function is the task of the state experiment stations, and to that end cooperative turf fungicide trials have been organized on a national basis. Criticism of some of the results may be justified when reliance is placed on natural presence of the disease and fungicide applications are not properly timed with fungus development. Admittedly, knowledge of the epidemiology and life history of turf pathogens is inadequate, but as it accumulates from research the curative as well as the protective action of fungicides may be better utilized.

In evaluating control measures for recommendation to others dependence

upon natural or chance inoculation and spread of the various pathogens is often disappointing and inefficient. This may be overcome by isolation of the fungus in pure culture and its growth in quantity on sterilized grain for spawn. At a time when favorable climatic conditions prevail, the infested grain is scattered evenly over the turf on which the chemicals are to be tested. The pre- or post-inoculation application of the fungicides will have a bearing on whether preventive or curative action is expected. The treated-inoculated areas are covered with strips or a blanket of burlap for 48 hours to maintain high humidity and to lower the grass carbohydrate reserves by shading. Opened sugar beet pulp bags sewn together are useful as a covering. Watering the burlap from one to three times a day may be necessary to maintain sufficient moisture, but the cooling effect of the evaporating water on the growth of the fungus must be allowed for. Disease damage is estimated from the percent of turf area affected or by a numerical index of severity. The randomized untreated check plots should exhibit 25 to 75 percent or more injury before the relative efficacy of a fungicide may be judged significant.

The limitations imposed upon some investigators by the necessity for using turf on greens in play have been a handicap. Untried materials that might injure are taboo, severe disease cannot be induced by spawning to provide a rugged test, and often only one type of grass is present. Experimental areas purposely grown by greenkeepers' research institutes or state experimental stations are the answer. In Rhode Island, experience has proved that variation between grasses in susceptibility to fungi and to chemical injury make it advisable to use for tests 2 to 4 kinds planted in adjoining uniform blocks in order to avoid incorrect conclusions. Treatments can be so clear cut on turf that 5 randomized 5′ x 5′ plots (25 sq. ft. each) are productive of meaningful results. The data thus obtained are capable of statistical analysis for significance.

It is not generally realized that a greater variety of factors affect the performance of turf fungicides than comparable materials applied to vegetable and fruit crops. On turf, in addition to the washing and deterioration of residues, we found that some chemicals became biologically inactive due to reaction with the soil, sod, or extruded by-products from the grass blades themselves. The erratic behavior of some fungicides, such as phenyl mercurials, was found to be caused by chemical combinations with soil in dirty spray water or when leaves were covered with dust. Grass clippings will also inactivate the fungicidal action of some products. A common error is to cut the grass soon after a protective fungicide is applied—but it does happen!

A neglected factor in turf health is guttated water or so-called "dew." It is the droplets that appear at the tips of leaf blades, particularly during periods of cool, calm nights and warm days. Water is absorbed by the roots and moves to the blade ends faster than removed by evaporation. There it provides the moisture necessary for the rapid growth of fungus mycelium. During its

passage through the veins a growth by-product, glutamine, is extruded into the sap stream by the surrounding cells. As the drops of guttated water evaporate, this metabolite accumulates as a white deposit. This may cause injury to tender tissues at high concentrations (i.e., tip burn of seedlings), and thus provide a favorable start for infection by some weak pathogens through the injured tissues. Glutamine may serve as a nutrient for fungus growth and also inactivate certain fungicidal chemicals. Early morning watering of the greens causes the droplets to be dissipated and injury avoided. Likewise, poling or dragging a mat across the green accomplishes the same purpose.

Some difference of opinion exists as to the volume of spray necessary to achieve complete and uniform coverage. Since the prime object is to distribute a given small dosage of pesticide evenly, the amount of water used serves only as a diluent to facilitate spreading and may vary depending upon the application equipment. Ten gallons per 1,000 sq. ft. is being commonly used, for at such dilution the chance of "burning" is minimized and there is sufficient liquid to insure even distribution. Some find 5 gallons satisfactory with standard nozzles, as long as an equivalent amount of toxicant is applied to the same area. Using modern low pressure, microjet nozzles, a good job can be done for water-soluble pesticides with as little as one quart of liquid for 1,000 sq. ft. The controversy as to whether the fungicide should be "washed in" or not is resolved by understanding whether the pathogen is on the foliage or on the crown and roots, and also, whether the action desired is prevention or cure. For example, protection against the formation of mycelia by brown patch fungus is expedited by washing the fungicide from the foliage into the turf. To cure, however, by destroying the active fungus on the leaf blades in the enlarging "smoke rings", the fungicide should not be washed down after application. A dust can be applied to wet grass with satisfactory results and with less time and labor, but some are hazardous to the operator.

Most important turf disease fungi produce a resting propagative organ capable of withstanding unfavorable growing conditions and known as a sclerotium. It is a round, oval or flake-like compact mass of threads or hyphae surrounded by a dark-colored protective rind or layer of modified cells. They may lie dormant for weeks, months or years in the sod and are more resistant to injury from chemicals than are the grasses. Sclerotia of the brown patch, dollar spot, copper spot, snow scald, etc., pathogens each appear to have an optimum temperature and moisture range at which hyphae grow. This is the stage when the preventive fungicides are most effective. For example, the sclerotia of the brown patch fungus, *Pellicularia filamentosa,* germinate at about 64°-68°F. to form short hyphae a few millimeters long. Calomel, acting in the vapor phase, will diffuse through the sod and kill them. If left untreated and the temperature rises to above 80°F. with abundant moisture, the short mold threads rapidly elongate up and over the grass blades as a "smoke ring." Soon the attacked leaves wilt and treatment is too late. This

is the time to apply a mixture of mercury chlorides to the leaves; the corrosive sublimate component is the quicker acting and momentarily most useful.

Available information is sufficient on many diseases barely to outline points of interest. When this scattered material is compiled and presented, interested persons may be able to note the many grass troubles more accurately. A summary of pertinent literature has seemed advisable as a base for reporting the observations, experience and research data from investigations at the Rhode Island Station. As will be evident, knowledge of the fungi attacking turf has in varying degree progressed through the traditional stages of observation, description and classification prior to fundamental physiological and biochemical studies of the relationship between host and attacking pathogen. Such a digest as this can never be final or complete; it is merely a convenient marker along the advance of knowledge. It is expected that some will use it as a "working notebook" and make additions and corrections as observations present themselves.

Choice of name for each disease and pathogen has been based on common usage and judgment of viewpoints expressed in authoritative literature, appropriateness to major symptom and personal experience. The description of symptoms, supplemented by the colored illustrations, is to facilitate recognition by greenkeepers and lawnsmen of the more important diseases. While a "key" to aid in diagnosis is possible for some diseases, symptoms so overlap and are so modified by host and environment that its value is limited. The technical description of the fungi is provided to permit exact microscopic identification of the causal organism by the specialist without an extensive literature search. The greenkeeper, especially, will find a lens that will magnify 10x or 14x an excellent investment. It will help him to determine more accurately what is killing the grass and thus to use an effective control measure, where known.

Reported host grasses are listed for most diseases to assist in identification. Bentgrasses, fescues and bluegrasses make up the bulk of cultivated turf. However, to broaden the uefulness of this treatise, the ryegrasses, Bermuda grass, and the "weed" crabgrasses and wheatgrasses are considered, since they are often found in fairways, parks, cemeteries, and lawns. Hitchcock (54), *"Manual of the Grasses of the United States"*, has been followed for names of hosts, where possible.

A provisional "Turf Disease Control Timetable" has been prepared for the guidance of informed greenkeepers, which indicates the time when major diseases may be expected, the time and/or interval between fungicide applications, and the chemicals found most effective under Rhode Island test conditions. However, diseases may appear and cause considerable damage at times other than those indicated as the treatment dates. It is imperative to recognize the various diseases in order to use the "Timetable", and also to be able to apply immediately an effective fungicide to control unseasonable disease development.

DISEASE CONTROL TIMETABLE FOR RHODE ISLAND

Disease	*Time of Occurrence*	*Usual Air Temp. — °F.*	*Fungicide*[1]	*No. of Applications*	*Timing Interval (days)*
Brown patch	July-Sept.				
Sclerotia germinate		64-68	Mercury chlorides	4-6	7-14
			Thiuram	4-6	7-14
Foliage killed		75-89	Benzoquinone	4-6	7-14
Dollar spot	June-Sept.	64-86	Cadmium compounds	3	30
			Phenyl mercury compounds	5	14
Snow molds	Dec.-Apr.	41-59	Mercury chlorides	1-(2)	Dec. (Feb.)
			Thiuram	1-(2)	Dec. (Feb.)
Red Thread	May-June	64-72	Cadmium compounds	2	May, Sept.
	Sept.	26-39	Phenyl mercury compounds	3	May, Sept.
Copper spot	July-Aug.	61-86	Cadmium compounds	2	30
			Phenyl mercury compounds	4	14
			Mercury chlorides	4	14
Helminthosporium					
Bluegrass leaf spot		55-65		1	
Brown blight	Apr.-May Sept.		Phenyl mercury compounds		
Others	May-Sept.	70-85			
Pythium blight					
Grease spot	July-Aug.	90-95	Zineb; Zerbam	1-2	7-10
Others	Apr.-May Sept.-Oct.	77-86		1	

[1] Directions for use of fungicide are now required by Federal law on manufacturer's label. Dosage varies with type and content of active chemical ingredient which must appear on the package.

See "Control" under each specific disease for details, where known.

DISEASE-DIAGNOSIS

It is imperative that injuries be correctly identified in order that intelligent preventive or curative measures be applied. To that end descriptions of diseases caused by fungus pathogens are given in detail.

BROWN PATCH (Summer Blight)

Symptoms: Injury typical "smoke ring" of grayish-black mycelium from one-half to 1½ inches wide delimits the patch when the fungus is actively growing. The leaves become water-soaked, blacken, then collapse, dry and become light brown in color. The crown and roots of the grass plant are not generally killed. (See colored plates.) The fungus spreads from a central point leaving roughly circular wilted brown areas usually from 1 in. to 3 ft. in diameter, but may reach 20 ft. across (53).

Distribution: General where warm temperatures and high humidity prevail in the U.S.A., Canada, Europe, Africa, Japan, Australia and New Zealand.

Hosts: Agropyron cristatum, A. repens, A. subsecundum, Agrostis alba, A. exarata, A. palustris, A. stolonifera, A. tenuis, Festuca arundinacea, F. ovina var. duriuscula, F. rubra var. commutata, Lolium multiflorum, L. perenne, Poa ampla, P. annua, P. compressa, P. pratensis, P. trivialis. Tilford (114) reported *Poa pratensis* and *Cynodon dactylon* immune, but the latter was cited as more resistant in Queensland (3) than species of *Agrostis*, and susceptible by Monteith and Dahl (78), and by Ryker (90) in Louisiana.

Pathogen: Pellicularia filamentosa (Pat.) Rogers *(Rhizoctonia solani).*

Fructification resupinate, forming a delicate separable, flaky or thin-hypochnoid pellicle, when dry white to buffy, or semi-translucent; hyphae branching at right angles and with some cruciform cells, without clamps, 4.5 — 14 [—17] u in diameter, the basal long-celled and often somewhat colored, the subbasidial colorless, short-celled, with often barrel-shaped segments, branching abundantly and bearing the basidia in small imperfectly symmetrical cymes; basidia subcylindric and barrel-shaped (widest toward the middle) or obpyriform or clavate (widest at the summit), [10] — 12 to 18 — [23] by 8 to 11 — [12.5] u; bearing 4 sterigmata which arise as blunt knobs and later become horn-shaped, [3] — 5.5 to 12 — [20] by 1.5 to 3.5 — [4.5] u; spores ellipsoid or oblong ellipsoid, thin-walled, flattened on the inside, a little the broadest below the middle, truncate-apiculate, 7 to 12.5 by 4 to 7 u, occasionally germinating by a stout promycelium on which is borne a similar secondary spore (89). The sclerotia are generally dark brown, membranous, and about 2 mm. in diameter.

Epidemiology: Brown patch was recognized in a turf garden near Philadelphia in 1914 and a causal fungus isolated in 1915 (78). Piper and Coe (85) in 1919 and Oakley (82) in 1924 again called attention to the disease before Monteith (74) began the study of the problem of control.

Experienced greenkeepers often speak of "brown patch weather" and Dickinson (26) has made perhaps the most significant contribution toward more exactly delimiting the conditions. He used field, laboratory and greenhouse data to substantiate his findings. Inoculum in the form of sclerotia is

prevalent in most soils, hence outbreaks of the disease are dependent upon the presence of a susceptible host and favorable climatic conditions. The sclerotia, or resting bodies of the fungus, are on the soil surface and the grass where air temperatures can have a great influence on their development. Attacks have been observed on "dry" grass also indicating that air temperature may be a greater factor than free surface moisture. Brown patch is not necessarily nocturnal although best observed in early morning. It can be found in the daytime, particularly when turf is artificially shaded. However, at nightfall air temperatures often decline from 80°-95°F. to 60°-70°F. and dew, guttated water, or fog is produced which favors the development of the pathogen. Late afternoon watering may accentuate the effect, since moisture may be more important as a modifier of air temperature than a direct factor for sclerotium germination. Two stages in the fungus development occur: first, short mycelia (10 to 15 mm.) grow from the sclerotia and are dependent upon it for food; second, the short mycelia grow into independent, parasitic hyphae which become pathogenic to grass at about 73°F. and cease at about 90°F. If the air temperature is rapidly lowered to 64°-66°F. (17.5°-20°C.) as by a sudden cooling wind or rainstorm, the sclerotia may form the short mycelia within one-half to one hour. Then as the temperature rises the disease may become evident within 3 hours. The short mycelia are destroyed or retarded below 17.5°C. and at 35°C., or higher. If the favorable temperature for sclerotial germination (64°-68°F.) is reached from a lowered temperature, 8 to 10 hours may elapse before the short mycelia will develop. Dickinson (26) demonstrated with pots of Metropolitan bentgrass inoculated with sclerotia that 60 percent would form short mycelia within 45 minutes when cooled to 64° to 68°F. while none kept at a constant temperature germinated within the same period. Since the favorable lowering and subsequent rise in temperature occurred only about 5 percent of the time during the brown patch season in Massachusetts, Dickinson (26) points out that cognizance of these fluctuations will permit more efficient timing of fungicide applications. However, Dahl (21) states that, based on 5 years of observations at Arlington, Va., temporary lowering of temperature did not stimulate sclerotial germination. He found that on 82 percent of the days when brown patch occurred, the minimum temperature was above 21°C. Sclerotia of some strains germinated at 8°C., all did so between 12° and 36°C. (most rapidly from 28° to 32°C.), but none at 40°C.

Pathogenicity of the *Rhizoctonia* or sclerotial stage of the fungus varies considerably with races (isolates) and soil type according to Sanford (94). Isolates of *Rhizoctonia* from grass injured by brown patch and from potatoes were compared by Monteith and Dahl (77). They found that morphological and physiological differences were too variable to be diagnostic. The brown patch strain grew more rapidly at higher temperatures (25° and 30°C.) than

did the potato strains. Van Luijk (116) found his isolate of the fungus to be most pathogenic at temperatures between 24° and 32°C.

Blair (13) observed that a neutral soil reaction appears optimal, but the fungus grows within variation from pH 5.8 to 8.1. Likewise, Hearn (53) found the disease in Texas on soils ranging from pH 6.0 to 8.2. This is contrary to some casual reports that the disease is most prevalent on wet, acid soils. Monteith and Dahl (78) found that the fungus would grow between pH 3.6 and pH 8.2, with the optimum between pH 5.8 and 7.0. Therefore, they point out that the disease can not be controlled by modifying the soil reaction, since the fungus has a wider range of acidity and alkalinity for growth than the bentgrasses and the optima for both grass and pathogen are about the same.

Wernham (121) reported that 10 lbs. of hydrated lime per 1,000 sq. ft. applied every 2 weeks gave superior control to Spergon at the rate of 3 or 6 oz., Thiosan (thiuram) at 4½ oz., Caloclor at 3 oz., or Semesan at 4 oz. per 1,000 sq. ft. Blair (13) found mycelial growth greater at 30 and less at 80 percent soil saturation, presumably due to decrease in soil aeration with increased soil moisture. His experiments indicate that the fungus may derive nutrients from the soil providing a sufficient food potential is present for saprophytic growth. Sclerotia may be viable 4 to 5 years in turf or on soil. The antagonism of common soil fungi to the brown patch fungus has been confirmed by Jaarsveld (57). Observations at Rhode Island indicate that mowed or cut turf is more readily attacked than uncut seedlings. An excess of nitrogen, especially as ammonium sulfate, appears to predispose the grasses to attack.

Control: Despite the trial of many fungicidal chemicals for control of this disease, the mercury chlorides, i.e., calomel and corrosive sublimate, usually in combination, still are favorites. Monteith (74) and Monteith and Harmon (79) did the pioneer work with mercurials and concluded that the degree of control is largely dependent upon the amount of mercury present. Other inorganic mercury salts and proprietary organic mercury compounds have been tried with success by Hearn (53) and many others, but they have not come into general use largely due to greater cost. Recently Keil and Howard (61) have shown that water soluble phenyl mercury acetate and lactate are effective at dilutions of 1:16,000.

Harrington (51) reported that thiuram applied weekly in dry sand at 4 oz. per 1,000 sq. ft. and watered in lightly gave complete control during the summer compared to 70 percent disease in check plots. A wettable form (Tersan) is now generally applied as a spray, but control has not always been complete (121).

Bordeaux mixture and copper sulfate (blue vitriol) alone or combined with potassium permanganate (3) have been used. However, Tilford (114), Monteith and Harmon (79), and Monteith and Dahl (78) point out that accumulation of copper is very harmful to grass roots. Cadmium compounds

do not appear to be effective against this disease. Mechanical disturbance by poling was believed by Dickinson (26) to kill easily the short sclerotial mycelia. The authors believe that the advantage of this treatment is to disperse the water droplets on the grass blades and thus inhibit the fungus attack. Complete control may not always follow early morning poling because favorable conditions for growth may occur in daytime as well as nighttime.

DOLLAR SPOT

Symptoms: Injured areas at first brown, later bleached straw-colored, about 2 inches in diameter; then severe coalescing to form large irregular patches. The "dollar spots" persist, if uncontrolled, for many weeks or months despite mowing. When leaves are being killed a fine, white, cobwebby growth of mycelium may be observed in the early morning while dew is still present. (See colored plate.)

Distribution: General in the U.S.A., Canada, Britain, Holland, Australia.

Hosts: Agrostis spp. (especially *A. stolonifera, A. palustris*), *Festuca* spp. (*F. ovina* var. *duriuscula*), *Poa* spp.

Pathogen: Sclerotinia homoeocarpa F. T. Bennett.

Bennett (12) points out that several strains exist which maintain their respective characteristics under natural and cultural conditions. They differ in capacity for spore production; one yields ascospores and conidia, another ascospores only, and others reduced sporiferous structures only. Mycelium at first white, wooly; later becoming cinnamon to reddish-brown, chalcedony yellow, or faintly tinted bluish-green depending upon the strain. Sclerotia black, small flakes to expansive parchment-like membranes; formed by conversion of superficial hyphae of the white mycelium into a mosaic of small thick-walled cells. Conidial fructification cupulate, 0.4 to 0.6 mm. in diameter, light brown; rim dark-colored, irregular; stalk 4 to 6 mm. long, pale cinnamon. Conidia borne singly on hyphae within the cup, hyaline, 1-septate, constricted at the septum, apex either rounded or narrowing to a blunt point; 15.6 to 20.8 by 4.5 to 5.2 u, commonly 20 to 21 by 4.6 u. Microconidia (if formed) spherical, hyaline, 1.5 to 2. u, in minute cream-colored pustules; not known to germinate. Apothecia cupulate 0.5 to 0.8 mm. to disc- or funnel-shaped 1. to 1.5 mm. in diameter, pale cinnamon to dark brown, with prosenchymatic exciple, arising from micro-sclerotia or expansive sclerotial flakes or membranes; stalk cylindrical, slender, flexuous, from 5 to 10 mm. or more long, arising singly or in clusters, simple or branched in the upper part. Asci cylindro-clavate, inoperculate, 140 to 170 by 10.4 to 11.5 u commonly 150 to 165 by 10.4 u. Ascospores 8, uniseriate, hyaline, oblong-elliptical, bi-guttulate, unicellular, a delicate median septum often forms during germination, 15.6 to 16.9 by 5.2 to 6.5 commonly 16 by 5.5 u, germtube grows from the end of each cell. Paraphyses few, cylindro-clavate, sparsely septate, 80 to 120 by 2.0 to 2.2 u. (12).

Epidemiology: Sporophores have not been found in nature but knowledge of their existence and probable time and conditions for their formation should be helpful in the search. The fruiting bodies were developed during April and May at room temperature by cultures 5 to 15 months old (12). The nature of the substrate and the particular isolate had a marked influence upon the capacity of the fungus to produce spores.

Conidia have been found to germinate in water at 15° to 20°C. within the pH range 4 to 8, but most vigorously between pH 5 to 7. Ascospores were likewise germinated by Bennett (12) within this pH range, except at 10° to

12°C. which probably more nearly corresponds to the temperatures prevailing when they are discharged. After investigating mycelial growth he concludes (12) that acid conditions are more favorable to rate and vigor than alkaline conditions, but there was no marked difference within the range of pH 4 to 7. Any influence the acidity of the soil may have on the development of the disease is likely to be by indirectly influencing the health of the host plant only.

The effect of temperature on rate of mycelial growth appears (12) to vary with the isolate (strain). Some were found to grow very slowly at 0°C. while others not at all; some reach an optimum at 20°C., others 25°C., and still others 30°C.; some are devitalized at 35°C. within 2 days, while others can withstand 37°C. for at least 7 days. Dollar spot may become active when soil surface temperatures reach a minimum of 60°F. in Rhode Island and continue to spread to a maximum of injury when 80°F. is reached, depending chiefly upon hours of rainfall and high humidity. It is most noticeable when the grass is not growing rapidly due to low soil moisture, yet when there is sufficient surface moisture from dew, fog, and sprinkling to permit the pathogen to develop.

Control: Cadmium-containing fungicides (Cadminate, Puraturf 177, and Crag 531) appear, on the basis of Rhode Island and national tests, to be the most specific and safe agents for both the prevention and cure of this disease. Research data indicate that areas of turf having as much as 80 percent of the foliage killed by the pathogen may be reduced to less than 20 percent infection within 15 days after a single application and to zero infection within 30 days where a second application was made at the 15-day interval. Remarkable residual protection has been demonstrated. Monthly applications during the growing season are generally sufficient. The cadmium-containing products are potent, requiring only 0.3 oz. of actual cadmium per 1,000 sq. ft., yet massive overdoses have failed to cause appreciable turf injury. They are reported to have a much lower toxicity for people and animals than mercurials.

Organic mercurials (Puraturf, PMAS) and inorganic mercury mixtures of calomel and corrosive sublimate are effective preventives, but are more prone to cause injury. Thiuram (Thiosan) was reported by Harrington (51) as providing control when applied at 2 oz. per 1,000 sq. ft. at weekly intervals in dry sand and watered in lightly. Trials at Rhode Island and some other experiment stations, where the disease is severe, have not given entirely satisfactory results. Tervet (113) applied 1 lb. (Thiosan, 50% active) to 6,000 sq. ft. of turf at 2-week intervals in top dressing or as a spray and obtained good protection against dollar spot. However, even 2 lbs. or 3 lbs. per 6,000 sq. ft. did not give control after the disease was established. He suggests that the variable success obtained is due to too heavy watering after application. Bordeaux mixture has been tried without success for control of this disease.

PINK SNOW MOLD

Symptoms: Injury characterized by salmon-pink spore masses (sporodochia) in minute, gelatinous heaps on dying leaves. First appears as roughly circular patches 1 to 2 inches in diameter with a fringe of white or pinkish mycelium and spores; then may enlarge to 12 inches across and coalesce to cover large areas. The fungus may grow beneath the snow cover, more commonly just as the snow melts, or during cold winter rains.

Distribution: General in northern U.S.A., Canada, and Europe.

Hosts: Agropyron dasystachyum, A. elongatum, A. inerme, A. intermedium, A. michnoi, A. repens, A. semicostatum, A. sibericum, A. spicatum, A. subsecundum, A. trachycaulum, A. trichophorum, Agrostis alba, A. canina, A. palustris, A. stolonifera, A. tenuis, Cynodon dactylon, Festuca arundinacea, F. elatior, F. idahoensis, F. megalura, F. ovina, F. rubra, F. rubra var. *commutata, Lolium perenne, Poa ampla, P. annua, P. arctica, P. bulbosa, P. cusickii, P. leptocoma, P. palustris, P. pratensis, P. secunda.* Reports differ on relative susceptibility, but it seems evident that Colonial, Washington, and Metropolitan bents are more resistant than Seaside.

Pathogen: Calonectria nivalis (conidial stage *Fusarium nivale* Auct.) Schaffnit.

Aerial mycelium spiderweb-like, clear, faint pink to rosy, branched, filamentous, loose or tufted; bearing scattered, small powder-like masses or balls of conidia, when moist slimy, rosy-white to salmon-orange color, upon drying darkening to brick-red or cinnamon-brown. Macroconidia normally spindle- or sickle-shaped, curved, tapering at each end, bluntly conical to rounded off, heel-less, 1- or 3-septate, few intermingled nonseptate and 4- to 7-septate forms; 1-septate, [9] — 13 to 18 — [23] by [2.2] — 2.4 to 3 — [4.5] u, 3-septate [13] — 19 to 27 — [36] by [2.3] — 2.8 to 3.8 — [4.5] u. Chlamydospores and sclerotia absent. Stromata delicate, thin, transitional or plectenchymatous, wrinkled, clear isabelline, rosy, orange- to brick-red, later chestnut-brown.

Perithecia always formed upon wrinkled, tuberculate stroma, singly or in groups, black, bulbiform to oval, averaging 0.16 (0.08 to 1.1) mm. in diameter, fragile peridium formed by 3 plectenchymatic cell layers uniting. Ascospores arranged uniseriately or indistinctly biseriately, 8 to the ascus; spindle-form, straight or curved, smooth, 1- to 3-septate, [10] — 13 to 18 — [20] by 3.4 — [4] u. (125).

Epidemiology: Accounts in the literature based on field observations are not always clear as to the pathogen responsible and hence some discrepancy may occur. Dahl (20, 22) observed that the disease was promoted by a damp autumn, snow falling on unfrozen ground, deep snow, and a prolonged, cold, wet spring. He also noted that fertilization, particularly organic manures, in late autumn and covering the greens with straw late in the season favored the disease. Likewise, Tyson (115) reports that severe infection occurred where organic nitrogen fertilizers, ammonium sulfate, urea, and sodium nitrate were used, but less infection with calcium nitrate. The suggestion was made by Jones (59) that the disease is more prevalent on acid soils. Yet, Schaffnit and Meyer-Hermann (95) found their strain of *Calonectria nivalis* (*graminicola*) to grow best at pH 7.9; being unable to grow in acid soils. Although the conidia did not germinate below pH 5.0, *F. nivale* was found by Bennett

(10) to grow over a wide range of hydrogen-ion concentration (pH 2.5 to 13.) with the optimum about pH 6.6 to 6.9. He concludes that the British strain can exist and attack (cereals) in all normal field soils whether of acid or alkaline reaction. However, since the fungus is not able to attack vigorously growing tissues, under good growing conditions no serious damage occurs.

When *Fusarium nivale* is the sole or the dominant organism present, Bennett (10) found that the aerial mycelium is freely and rapidly developed when the affected turf is placed in a moist chamber in a warm room. The pathogen persists as dark-brown, plectenchymatic aggregations of mycelium embedded in plant residues. Although mycelium can withstand temperatures as low as —20°C. and makes definite growth at 0° to 1°C., it grows most vigorously at 20° to 21°C. and ceases growth at 32° to 33°C. He concludes (10) that the natural meteorological conditions most favorable for all phases of *F. nivale* are those of a moist summer, but injury is more obvious as a winter disease owing to lack of new growth of the grasses. As the result of field observations, Dobrozrakova (27) believes that the identity of the species of *Fusarium* that cause winter injury (to cereals) is closely dependent on the meteorological conditions of each individual year. She found (27) perithecia in fields near Leningrad at the beginning of July which were pinkish, later light brown; yet those formed in pure culture on *Melilotus* stems were black. Perithecia may appear in cultures at room temperature from July to September when the fungus is isolated from diseased patches. Bennett (10) found wheat meal agar to provide for the best growth of the fungus.

Control: The most satisfactory treatment appears to be the application of a calomel-corrosive sublimate mixture at the rate of 2 to 4 oz. per 1,000 sq. ft. before the first snow is forecast and again, where possible, during a midwinter thaw. Red mercuric oxide has also been used at the same rate. Wettable thiuram (Tersan) is being sold as a control agent. Bennett (9) in England has suggested a combination of malachite green (1:20,000) and Bordeaux mixture.

SNOW SCALD (Winter Scald)

Symptoms: The injury takes place both under the snow and as the snow is melting; starting from a roughly circular area 1 to 2 inches in diameter the fungus spreads outward as a band or halo of white, blue-gray, or almost black mycelium about 1 inch wide. Usually damage stops when the area reaches 1 to 2 ft. in diameter due to drying out or warming up of the turf surface. However, patches up to 20 ft. in diameter have been recorded.

Distribution: General in northern U.S.A., Canada, Japan, northern Europe.

Hosts: Agropyron spicatum, Agrostis palustris, A. stolonifera, A. tenuis, (A. canina is resistant), *Festuca rubra, Lolium perenne, Poa annua, P. palustris, P. pratensis, P. secunda, P. trivialis.*

Pathogens: Typhula itoana Imai, *T. idahoensis* Remsberg.

Sclerotia tawny to hazel-brown, immersed-erumpent, frequently falling away from substrate, single, or coalesced, smooth at first, rough when dry or old, spherical to subglobose, often somewhat flattened or concave below and convex above, 0.5 to 2.0 by 1.5 to 4 mm.; rind golden to reddish-brown, about 7.7 u thick, tough, composed of a smooth gelatinous layer on the outer walls of enlarged peripheral cells; medulla paraplectenchymatous, center often prosoplectenchymatous with hyphae loosely interwoven, small sclerotia solid, large ones and coalesced masses often hollow. Sporophores clavate, erect, straight or slightly curved, apex broad, round or slightly pointed, tapering into stipe, simple or branched, one or more (usually one) arising from each sclerotium, often slightly pubescent at the base, 8 to 25 mm. tall, clavula rose color, opaque, stipe white, translucent, becoming flesh-color to bittersweet-pink when dry; clavula straight, erect, smooth, cylindrical or somewhat broadened and flattened, tapering toward a round or slightly pointed apex, 10 to 15 mm. tall, 1 to 2 mm. broad, hydrangea-pink to pale vinaceous-pink, made up of a hollow cylinder of agglutinated hyphae bearing the hymenium which is continuous over the apex, crystals present in the hymenium and subhymenium; stipe distinct in size and color from the clavula, erect, straight, pubescent at the base with hairs clasping the sclerotium, 5 to 10 mm. long, 0.5 to 1.0 mm. in diameter, white, solid, hyphae often slightly incrusted with fine crystals; basidia broadly clavate, 27.2 to 34.6 u long, 5. to 7.8 u wide; basidiospores ovoid, flattened on one side above a pointed apiculus, 4.3 to 7.8 by 11.3 to 14.8 u, average 6 by 11.7 u. Mycelial growth (in culture) is abundant, white, webby, radiating, concentrically banded, and fan-shaped; sclerotia appear in 5 to 10 days. Hyphae septate, hyaline, clamp connections, profusely anastomose (88).

Typhula idahoensis causes a disease of grasses similar to *T. itoana* described above, but the always single sclerotia of the former can be distinguished by their chestnut-brown to bone-brown color when mature; rarely branched, fawn to wood-brown sporophores 5 to 10 mm. tall.

Epidemiology: The sclerotia lie dormant in the field until late autumn when they may form fruitbodies bearing basidiospores (111). Under snow, especially when the ground is not frozen, mycelium is produced rather than fructifications due to reduced light, high humidity, and favorable temperature. The minimum temperature for growth appears to be at least —5 °C., the optimum between 8° and 15°C., and the maximum 22° to 23°C. (112). Growth is profuse at pH 7.0 and is inhibited at pH 3.5. Sclerotia can remain viable for 16 to 25 or more months. In the spring, the fungus forms sclerotia and becomes inactive as the soil warms up. It is not seed borne.

Control: The standard treatment is the use of mercury chlorides (calomel-corrosive sublimate mixtures). Noer (81), among others, reports satisfactory control by the use of calomel-corrosive sublimate mixture 2:1 (i.e., Calo-clor) at 3 to 4 oz. per 1,000 sq. ft. and thiuram (Thiosan) at 6 to 8 oz. per 1,000 sq. ft. in Wisconsin applied in November before the first snow was forecast. Considerable difference in susceptibility to the disease occurs among the various species and varieties of grasses. Phosphate fertilizers have been reported to decrease injury from *Typhula.*

FROST SCORCH (String of Pearls)

Symptoms: Scorched, withered leaves similar to frost damage, covered with rows of tiny sclerotia are typical injury (93). Davis (24) found 175 sclerotia per square inch formed on one diseased patch. Affected leaves curl

up, wilt, become bleached; leaf sheaths wither. Pape (84) described the conspicuous symptom as the bleached, dry, bent aspect of the rolled leaves and their termination in a long pale tendril, the basal portion remaining normally green and flat. The apex of the tendril on each leaf (except the lowest) is crooked and adheres to the rolled portion of the next leaf forming a kind of loop that imparts a peculiar concatenate aspect. The white or delicate gray mycelium extends along the midrib, producing a dry, pale stripe in the tissue of the green portion. Sclerotia are found on both the tendrils and flat portion of the blade.

Distribution: Northern North America, northern Europe.

Hosts: Agropyron subsecundum, Agrostis alba (reported resistant by Stout, (110)), *A. hiemalis, A. stolonifera, A. tenuis, Poa pratensis, P. trivialis.*

Pathogen: Sclerotium rhizodes Auers.

Sclerotia form in rows, bead-like, superficial on leaves, white when young, later brownish, becoming dark gray to black, oval to spherical, 1 to 5 mm. in diameter. Mycelium white in mass, septate, branched, no clamp connections, chiefly intercellular in leaf, stem, rhizome, or surface root. Baudys (8) suggested that *S. rhizodes* belonged in *Sclerotinia,* but spore-forming bodies have not been seen in nature or culture.

Epidemiology: Most common on poor, sour soil (109). Sclerotia develop within 10 to 20 days on the mycelium in infected leaves and germinate when 2 to 36 months of age. Although sclerotia that overwintered on the ground at Madison, Wisconsin, had not germinated by March 24, the disease developed in early April (110). The disease is most conspicuous during the April-May period of rapid growth of the host plant, but is checked on fully developed leaves during June and July. Each leaf as it unfolds may contain the fungus, but exposure to dry warm air stops the formation of mycelium and sclerotia. The mycelium is systemic and perennial (110); the majority, if not all, of the culms arising from the same rootstalk are infected. There is evidence that the fungus may be soil-inhabiting and penetrate roots directly from the soil.

Control: Collect clippings when mowing diseased areas to remove inoculum (sclerotia) on the leaf tips. Mercury-containing fungicides may be used, if serious.

WINTER CROWN ROT

A serious disease is reported from Canada by Cormack (19) caused by a low-temperature, unidentified basidiomycete. The coarse white mycelial mats have been found attacking *Agropyron repens, Agrostis alba, Festuca elatior, F. rubra,* and *Poa pratensis.* The plants become susceptible between late November and late December.

The reddish "threads" (stromata) of the fungus seem more diagnostic than the pinkish cast imparted to dead or dying grass. Therefore the name "red thread" commonly used in England appears preferable.

Symptoms: A pinkish web of hyphal threads attacks the leaves and leaf sheaths only, which become water-soaked at first, then lose their green color as the tissues dry out. Turf is killed in distinct, isolated patches 2 to 15 inches in diameter. Pink gelatinous branched strands or "horns" of the fungus develop during moist weather and tend to bind the leaves and stems together. They shrink on drying, become bright coral-pink in color, irregularly tapering, 5 to 50 mm. in length, and brittle fragments serve to disseminate the pathogen.

Distribution: General in U.S.A., New Zealand, England.

Hosts: Agropyron repens, A. scabrum, Agrostis alba, A. canina, A. palustris, A. stolonifera, A. tenuis, Festuca dertonensis, F. myuros, F. ovina, F. ovina var. *duriuscula, F. rubra, F. rubra* var. *commutata, Lolium perenne, Poa annua, P. pratensis.*

Pathogen: Corticium fuciforme (Berk.) Wakefield.

Hyphae tinged pink, with clamp connections, thin-walled and rather vacuolate, 2 to 4 u in diameter, walls somewhat gelatinous. Sterile papillae projecting above the hymenium but developed mostly where the hymenium is not formed. Hymenium delicate effused, somewhat gelatinous incrustation forming patches on leaves and stalks of grasses. Basidia slightly tinged pink, clavate, with 2 to 4 stout, curved sterigmata, 5.5 to 7 u in diameter. Basidiospores pip-shaped, one side depressed, hyaline, apiculate, 11 to 12.5 by 5 to 6 u (118).

Epidemiology: The pink vegetative stromata serve as inoculum to spread the pathogen according to Wakefield (118). Leaves and stems of both seedling and established turf are attacked. Bennett (11) reported that neither summer heat nor winter cold would kill the fungus, while Libby (67) found that dry stromata can survive 2 years of storage. They remained viable after storage 18 months at 25°C. for Erwin (33). Mycelial growth starts within 2 days from viable stromata under favorable conditions. With abundant moisture present Erwin (33) reports the cardinal temperatures for growth as 1°C. minimum, 18° to 22°C. optimum, and 30°C. maximum. Two outbreaks occurred on the turf plots of the Rhode Island Station in 1948; on June 10 the soil temperature 1 inch beneath the surface was 72°F. maximum and 66°F. minimum, and on September 22 it was 39°F. maximum and 26°F. minimum. Erwin (33) studied the growth of the pathogen extensively in culture on various media and its tolerance of acidity. While growth occurred between pH 3.6 and pH 8.0 the rate at a given pH level depended upon the medium. Beer wort agar at pH 4.9 permitted the optimum colony size of the 6 media he reports on. The amount and color (white to dark pink) of mycelium varied with the artificial substratum used for laboratory culture. In England the disease is reported as most prevalent where soils are chalky or sandy and having a pH range of 3.5 to 7.5.

Control: Cadmium compounds and mercurials both appear to give satisfactory control, especially when applied prior to infection. Malachite green (1:10,000) as a water solution was effective for Bennett (11) and Semesan at 1 lb. per 1,000 sq. ft. applied in sand was successful. Erwin (33) reports that a mixture of calomel and corrosive sublimate (2:1) gave good control when applied at 2-week intervals at the rate of 3 oz. in 20 qts. of dry sand per 1,000 sq. ft. and then watered. He did not find that hydrated lime applications prevented the disease as might be expected when the pH for its optimum growth is considered. The stromata (sclerotia) were killed after 72 hours exposure to mercury vapor without injury to grass blades (33). Injury from the fungus may be decreased by the application of nitrogenous fertilizers to low fertility turfs.

COPPER SPOT

Symptoms: Individual injured patches of turf 1 to 3 inches in diameter, coppery or orange colored (See color plate). Grass blades covered with characteristic salmon-pink spore masses (sporodochia).

Distribution: Louisiana, Pennsylvania, Rhode Island, California.

Hosts: Agrostis canina, A. stolonifera.

Pathogen: Gloeocercospora sorghi Bain & Edgerton.

Vegetative hyphae septate, hyaline, intramatrical, emerging as branched hyphae from stomata. Conidia unbranched, hyaline (salmon-pink in mass), many septate, elongate to filiform, 20 to 195 by 1.4 to 3.2 u (average 82.5 by 2.4 u), and borne in a slimy, pink matrix on hyaline, septate, simple or branched conidiophores, 5 to 10 u in length. Sclerotia black, lenticular to spherical, 0.1 to .2 mm. in diameter; formed in the necrotic host tissues, abundant (7).

Epidemiology: The fungus overwinters as sclerotia which resume vegetative growth in the spring after rainy periods when soil temperatures, 1 inch deep, reach a minimum of 62°F. for 7 days. The hyphae enter the grass blades through stomata, symptoms of injury appear within 24 hours, and spore masses are formed 24 to 48 hours later. The conidia germinate in water within 5 hours and serve to disseminate the pathogen during the growing season. In culture the optimum temperature for growth is about 28° to 30°C. The disease is most destructive to velvet bent growing in soil between pH 4.5 and 5.5. As lime is added and the acidity of the soil decreased, the disease incidence becomes less.

Control: Cadmium compounds, especially organic ones (Puraturf 177, Cadminate), have proved most effective in Rhode Island. Phenyl mercury compounds (Puraturf, PMAS) are also satisfactory.

HELMINTHOSPORIUM BLIGHTS

1. Bluegrass Leaf Spot

Symptoms: Scattered circular to elongate leaf spots, 0.5 to 3 by 1 to 8 mm., with prominent reddish-brown to black borders, at first dark to light brown in the center but becoming straw-colored to white. Also a foot-rot stage where the whole leaf sheath is affected, the stem and crown are killed, and brown lesions form on the white underground rhizomes.

Distribution: General in U.S.A. but most injurious in northeastern states, Britain.

Hosts: Poa arida, P. compressa, P. pratensis, P. secunda.

Pathogen: Helminthosporium vagans Drechsler.

Conidiophores emerging from stomata or between epidermal cells of tissues sometime after death, usually singly or less frequently in pairs; typically simple although occasionally branching; dark olivaceous; usually measuring 8 to 10 by 50 to 280 u; 1- to 10-septate, the septa occurring at intervals of 15 to 40 u; approximately straight up to point of attachment of first spore 40 to 150 u from base; successive spores produced at apices of moderate or often pronounced geniculations. Conidia dark olivaceous when fully mature; cylindrical or slightly tapering toward the hemispherical ends; measuring 17 to 23 by 25 to 130 u; 1- to 10- (usually 5 to 8) septate, the septa not associated with constrictions in the peripheral wall; the latter always thick and including the dark hilum within its contour (29).

Epidemiology: Favored by cool rainy weather; generally appears in early spring, sometimes in late fall. Footrot is most severe on close clipped turf.

Control: Horsfall (55) obtained control with copper-lime dust. Monteith and Dahl (78) suggest raising the height of cut or leaving uncut to mitigate damage from the destructive footrot.

2. Zonate Eyespot

Symptoms: Numerous yellowish or pale straw-colored leaf spots, 0.5 to 1 by 1 to 4 mm., elongated longitudinally, and with a narrow brown border. The spots are generally absent from the leaf sheath, and when numerous they become confluent on the lamina and thus cause somewhat extended dead areas (52). Leaf lesions are first visible as minute brown spots in the green tissue; increase in length and width, the center meantime fading to almost white or light straw-color, imparting an "eyespot" appearance. Under favorable conditions the fungus may occupy the entire width of the leaf blade for 1 cm. or more; dead areas usually show a number of roughly concentric brown markings, from successive spreading of the fungus into new zones in the presence of dew, and giving the areas a distinctive zonate aspect (29, 78).

Distribution: General over U.S.A.

Hosts: Agropyron elongatum, A. intermedium, A. repens, Agrostis canina, A. palustris, A. stolonifera, Cynodon dactylon, Digitaria ischaemum, D. sanguinalis, Festuca hookeriana, Poa pratensis.

Pathogen: Helminthosporium giganteum Heald and Wolf.

Conidiophores dark brown, many septate, 9 to 12 by 200 to 400 u, with a slightly bulbous base; spores elongated, cylindrical with slightly tapering ends, 5-septate, pale brown, densely granular contents, 15 to 21 by 300 to 315 u (29, 52). Live spores were found to be hyaline, colorless by Drechsler (29).

Epidemiology: Overwinters as dormant mycelium on old leaves; conidiophores and conidia are produced from late spring to September. Spores short-lived, seldom more than 2 weeks; produced in small numbers; germinate promptly in water. Injury occurs during warm, wet seasons particularly in August and September.

3. Fescue Netblotch

Symptoms: Infected green tissues show abundant dark-brown transverse discolorations resembling short pieces of dark thread across the leaf with darker lengthwise streaks forming a netlike pattern. Sometimes lesions are distinctly limited, dark-brown spots 5 to 28 by 1 to 1.5 mm.; leaves yellow and die back from the tips.

Distribution: General in U.S.A.

Hosts: Festuca arundinacea, F. elatior, F. occidentalis, F. rubra var. *commutata.*

Pathogen: Helminthosporium dictyoides Drechsler.

Sporophores dark brown or olivaceous, emerging singly or in groups of 2 to 6 from stomata or between epidermal cells, measuring usually 6 to 8 by 70 to 150 u; 3- to 6-septate, the septa generally occurring at intervals of 10 to 30 u; producing first spore usually at a distance of 50 to 100 u from base; points of attachment of successive spores marked by moderately or strongly pronounced geniculations. Conidia subhyaline and nearly colorless when newly proliferated, to yellow when fully matured; typically straight; maximum diameter usually at basal segment, 14 to 17 u; tapering uniformly and very perceptibly to apical segment, the latter in long spores frequently not exceeding 8 to 9 u in diameter, in short ones usually of greater diameter; more rarely approximately cylindrical, or short ellipsoidal. Length 23 to 115 u, usually 50 to 70 u, 1- to 7-septate, typically 3- to 5-septate, the septa not associated with perceptible constrictions except occasionally, and then constrictions most frequently present only at the basal septum; length of segments 7 to 24 u; typically 12 to 15 u. Contour of basal end hemispherical, of apical end hemispherical or hemi-ellipsoidal: peripheral wall or exospore uniformly thin, and entirely including the hilum within its contour.

Epidemiology: Common from early July until the end of August in the vicinity of New York City on *Festuca elatior* (29), but most active from December through early spring in Georgia (70).

4. Brown Blight

Symptoms: Leaves die back from numerous, dark chocolate brown, oval to longitudinally elongated spots, 0.1 to 0.3 by 0.2 to 1.0 mm., often coalescing. Withering begins at the blade tip and continues until the sheath is involved.

Distribution: General in U.S.A., Great Britain, Germany.

Hosts: Festuca elatior, Lolium multiflorum, L. perenne.

Pathogen: Pyrenophora lolii Dovaston (28); *Helminthosporium siccans* Drechsler.

Conidiophores olivaceous, emerging usually singly, less frequently in pairs, and rarely in groups of 3 from stomata or more especially from between epidermal cells on the vascular ridges; measuring 7 to 9 by 50 to 300 u; 1- to 9-septate, the septa inserted at intervals of 15 to 90 u; producing first conidium at a distance of 50 to 250 u from base; points of attachment of successive conidia at angles of geniculate irregularities occurring at intervals of 5 to 30 u. Conidia subhyaline or light fuliginous when newly proliferated, later becoming yellow, brownish, or brownish-olivaceous, never dark olivaceous like *H. sativum;* when fully mature provided with a moderately thick peripheral wall; typically straight or slightly curved; measuring 14 to 20 by 35 to 130 u; usually subcylindrical, or tapering slightly or more markedly toward apex, the distal segment often not exceeding 10 u in diameter, or rarely greater in diameter at the distal end than at the base. Apical and basal ends abruptly rounded off, the contours at the end being approximately hemispherical. Hilum moderately conspicuous included within contours of peripheral wall.

Perithecia irregular, 300 to 550 by 710 to 1,000 u in diameter with a short conical beak when mature; 1 to 50 flexuous, tapering multicellular, external setae, 8 to 11 u at the base and up to 200 u long. Asci 8-spored, 172 to 230 by 27 to 41 u, broadly cylindrical when mature, apex truncate with thickened apical ring. Ascospores pale brown, narrowly ellipsoid, 49 to 67 by 16 to 22 u, when mature usually have 5 transverse and 0 to 4 (typically 2) longitudinal septa. Spores germinate promptly by 1 to 5 germtubes and lesions develop within 4 to 5 days after inoculation. Aerial mycelium is light gray with olive-green plectenchyma; producing conidia after 3 weeks. Sclerotia numerous and large, bear flexuous tapering setae externally.

Epidemiology: Disease appears during cool, moist weather in early spring.

5. Leaf Mold

Symptoms: Indefinite; no pronounced discoloration; dry, withered leaves in fall.

Distribution: Oregon, Idaho, Iowa, Montana, New England states.

Hosts: Agrostis alba, A. scabra, A. stolonifera, A. tenuis.

Pathogen: Helminthosporium stenacrum Drechsler.

Conidiophores dark olivaceous, emerging singly or in pairs, usually between adjacent opidermal cells; measuring 7 to 10 by 80 to 250 u; 3- to 10-septate; the septa occurring at intervals of 10 to 35 u; producing the first spore usually 80 to 150 u from the base, the points of attachment of successive spores occurring at well defined geniculations. Spores subhyaline to yellowish when fully mature; 15 to 23 by 53 to 135 u; subcylindrical with hemispherical or hemiellipsoidal ends, or widest somewhat below the middle and tapering moderately toward the ends; the apical portion sometimes reduced to a somewhat narrowed distal prolongation; 1- to 11-septate, the septa not associated with constrictions or marked by barely perceptible constrictions. The peripheral wall thin and including the dark hilum within its contour.

6. Gray Leaf Mold

Symptoms: Leaf tips killed with vague lesions, gray mold appears on dead and dying leaves.

Distribution: Oregon, Washington, New York.

Hosts: Agrostis alba, A. exarata.

Pathogen: Helminthosporium triseptatum Drechsler.

Sporophores arising singly or in pairs, dark olivaceous, 6 to 8 by 200 to 400 u; usually 6- to 7-septate, the septa occurring at intervals of 18 to 40 u in length, averaging 25 u;

producing first spore approximately 175 u from base. Proliferation of spore associated with conspicuous local thickening of peripheral wall resembling ring or band, these thickenings occurring in series, giving upper portions of sporophore a more or less moniliform contour. Spores dark olivaceous, ellipsoidal or short cylindrical with hemispherical ends, sometimes tapering more or less toward basal end; regularly 2- to 3-septate, the septa not associated with constrictions in the peripheral wall; the latter unusually thick, although somewhat thinner at the distal end, and at the basal end diminishing markedly in thickness toward the hilum; the hilum not projecting beyond contour of proximal end; measuring 15 to 21 by 35 to 50 u; germinating by the production of 1 or 2 germtubes at positions adjacent to or in close proximity to hilum (29).

7. Red Leaf Spot

Symptoms: Numerous straw-color leaf spots with reddish-brown border, 0.5 to 2.5 mm. During prolonged periods of rain the spots may overlap, forming pseudozonate patterns. Spots surrounded by a water-soaked area, presenting a halo effect. Infected leaves wither and brown in dry weather.

Distribution: Eastern and middlewestern U.S.A.

Hosts: Agrostis alba, A. palustris, A. tenuis.

Pathogen: Helminthosporium erythrospilum Drechsler.

Conidiophores erect, brown or fuliginous, simple or occasionally somewhat branched, emerging singly or less often in pairs or larger groups mostly from between epidermal cells but sometimes from stomata, usually 6 to 9 by 75 to 275 u, producing the first conidium 75 to 125 u from the base and later conidia at variable successive intervals marked by geniculations; containing 1- to 10-septa that delimit segments mostly 15 to 35 u long. Conidia when mature distinctly yellowish, sometimes even light olivaceous; typically straight, rarely somewhat curved, mostly nearly cylindrical, the ends rather abruptly rounded off, containing 2- to 10-septa that are usually not marked by constrictions, but at times may be associated with perceptible constrictions; 8 to 16 u (average 12.3 u) by 25 to 105 u (average 65 u); individually including the nonprotruding hilum within the basal contour; germinating rather indiscriminately from any or all cells, but usually more abundantly from the thin-walled basal or apical cells than from the intermediate cells. Sclerotia produced in culture, minute, about 0.1 mm. in diameter (31).

Epidemiology: Spots appear after 16 hours of misty rain with warm temperatures.

8. Bermuda Grass Leaf Blotch

Symptoms: Effuse olive to fuliginous blotch on dry leaves.

Distribution: General in southern U.S.A., New York, Washington, D.C., California, Italy.

Host: Cynodon dactylon.

Pathogen: Helminthosporium cynodontis Marignoni.

Conidiophores dark brown, emerging singly or in pairs from stomata or between epidermal cells, show less tendency to occur in groups; 4 to 6 u by 50 to 150 u; 2- to 5-septate. Conidia 11 to 14 by 27 to 80 u, straight or more frequently somewhat curved, widest near the middle from which they taper slightly toward the abruptly rounded ends; subhyaline to fuliginous in color, never brown or dark olivaceous; 3- to 9-septate, the septa not being associated with constrictions in the relatively thin peripheral wall. They germinate promptly in water by two polar germtubes about 3 u in thickness, one from the apex and the other in immediate proximity to the hilum that can be distinguished within the contour of the rounded basal cell (29).

Other *Helminthosporium* leaf blights cause insufficient injury to warrant detailed accounts at this time. *H. dematioideum* Bub. & Wrob. has been re-

ported from the Atlantic states on *Agrostis alba* and *A. perennans* by Drechsler (29). *H. poae* Baudys has been observed in Oregon and Washington on *Poa secunda* and *P. trivialis* by Sprague (101, 102, 105), *H. tritici-repentis* Died. on *Agropyron repens* (47), *A. intermedium, A. smithii. H. sativum* was isolated from fescue in Georgia by Luttrell (70), while *Poa pratensis* is resistant.

MELTING-OUT (Curvularia Mold)

Symptoms: Dying out of turf; general leaf blight, seed rot, and seedling blight.

Distribution: General in northern plains of U.S.A., Canada, Europe, Australia, Java, India. Saprophytic on roots, culms, leaves, inflorescences of grasses and plant debris.

Hosts: Agrostis palustris, Festuca elatior, F. rubra, Poa annua.

Pathogens:

1. *Curvularia geniculata* (Tracy & Earle) Boedijn.

Mycelium at first white with scant aerial hyphae in radiating tufts or strands, growing 5 to 10 cm. in 10 days on malt extract agar, becoming dark olive-green to black as spores are produced. Sometimes hard, black, erect, cylindrical, branched stromata formed in cultures.

Conidia borne spirally or in whorls, brown unequally ventricose-fusiform, mostly 4-septate, the third cell from the base slightly larger and darker, the end cells nearly hyaline, more or less curved to nearly straight, mostly not strongly curved, [21] — 28 to 36 — [42] by 11 to 14 — [15] u. Conidiophores brown, septate, simple, geniculate at the tip, variable in length, [3] — 4 to 5 u in diameter (44).

2. *Curvularia inaequalis* (Shear) Boedijn.

Conidia borne spirally or in whorls, predominately 4-septate, occasionally 3- or 5-septate, [24]— 28 to 40 — [60] by [10] — 11 to 16 — [18] u, more strongly curved than *C. geniculata* and basal cell rounded rather than pointed (44).

3. *Curvularia lunata* (Wakker) Boedijn.

Mycelium dense bottle-green when grown in complete darkness; produces 2 alternate zones every 24 hours (white in darkness, pink in daylight) on 2 percent rice agar according to Nigam (80).

Conidia borne spirally or in whorls, pale brown, 3-septate, the third cell from the base larger and darker colored than the others, unequally ventricose-fusiform, more or less curved to nearly straight, [19.5] — 22 to 28 — [32] by [9] — 10 by 13 — [15.5] u. Conidiophores pale brown, septate, simple or branched, geniculate at the tip, variable in length, 3 to 5 u in diameter (44).

PYTHIUM BLIGHTS

1. Grease Spot (Spot Blight)

Symptoms: Injury a circular spot with characteristic greasy border (97) of blackened grass blades intertwined with a cottony growth of mycelium (78). The blackened host tissues become water-soaked as the pathogen penetrates, then wither, dry and become reddish-brown in color. Spots usually not more than 2 inches in diameter, but often in groups tending to form "streaks."

Distribution: Cosmopolitan throughout the world.

Hosts: Agrostis stolonifera, Festuca sp.

Pathogen: Pythium aphanidermatum (Edson) Fitzpatrick. (*P. butleri*)

Hyphae 2 to 8 u, chiefly 4 to 6 u, in diameter, hyaline, nonseptate except for fructification. Sporangia inflated, filamentous, branched or unbranched, length varying from 50 to 1,000 u plus by 4 to 20 u wide, usually forming complexes; zoospores reniform, 15 to 40 or more to a vesicle, laterally biciliate, measuring about 12 u long and 7.5 u wide. Oogonia spherical, terminal, rarely intercalary, measuring 22 to 27 u in diameter. Antheridia usually monoclinous, sometimes diclinous, typically intercalary though often terminal, 1 to 2 per oogonium, barrel- or dome-shaped, suborbicular, becoming cylindric or broadly clavate, usually measuring 9 to 11 by 10 to 14 u, producing a conspicuous penetration tube. Oospores aplerotic, single, with a moderately thick wall, measuring 17 to 19 u in diameter; germination by a germtube (72, 73).

Epidemiology: Monteith (76) points out that this disease is most destructive in hot (35°C. or 90° to 95°F.), damp weather, on poorly drained greens. Middleton (73) presents data showing little growth in 24 hours below 10°C., optimum at 34°C. and a trace at 46°C. This species is capable of growing at higher temperatures than any other member of the genus. The geographical-climatic source of the fungus does not appear to determine its ability to develop at a given temperature.

Control: Mercuric chloride applied as a spray was successful for Monteith (76). Iron sulphate applied at the rate of one pound in 30 gallons of water to 5,000 sq. ft. is reported to stop the disease.

2. Seedling Blight

Symptoms: Seedlings turn pale green, yellow, wilt and dry up.

Distribution: General, particularly in northern plains of the U.S.A.

Hosts: Agropyron spp., *Festuca* spp.

Pathogen: Pythium arrhenomanes Drechsler.

Hyphae inter- and intra-cellular, 2 to 5.5 u in diameter. Zoosporangia lobulate, composed of inflated communicating elements up to 20 u or more in diameter, often occurring in extensive complexes; evacuation tube 3 to 4 by 50 to 75 u; 20 to 50 zoospores developed in a vesicle. Zoospores biciliate, motile, later subspherical to ellipsoid, about 12 u in diameter, usually germinating by a single tube 2.5 to 3 u in diameter. Antheridia crook-necked, borne terminally or rarely laterally on branches arising from several (4 to 8) hyphae, each of which contributes up to 4, numerous, from 15 to 20 often visible in relation to an oogonium, 6 to 9 u in distal expanded portion and 12 to 25 u in length from apex to basal septum along the curved axis. Oogonia subspherical, terminal, occasionally intercalary, 24 to 35 u in diameter with wall about 0.5 u thick. Oospores subspherical, yellowish, sometimes completely filling the oogonium, 22 to 33 u in diameter with wall 1.2 to 2 u in thickness (30, 104).

Epidemiology: Middleton's (73) data for 24 hours growth show a trace at 4°C., optimum about 28°C., and little above 40°C.

Control: Fall seeding is preferred to spring seeding to avoid the disease. Seedlings should be at least 6 weeks old during the critical May-June blight period. The suggestion is made by Sprague (104) that an application of ammonium phosphate at 100 lbs. per acre will aid in control. Graham and Greenberg (40) found that the fungus cannot grow in the presence of 10 ppm. salicylic aldehyde, but the accumulation of the chemical in the soil predisposed wheat to attack.

3. Seed Rot and Damping-Off

Symptoms: Seed rots in the soil just after the seed coat is broken. Seedlings appear water-soaked, blackened, then shrivel and turn brown. Tissues rot and collapse just at or above the ground surface. Patches up to 4 inches in diameter are affected. In older plants infected rootlets turn brown and progressively die backwards.

Distribution: Cosmopolitan.

Hosts: Agropyron repens, Agrostis spp., *Festuca* spp., *Lolium* spp., *Poa* spp.

Pathogens: Pythium debaryanum Hesse, and *P. ultimum* Trow.

The two species are similar, except that the antheridium of *P. debaryanum* never originates immediately adjacent to the oogonium and is never swollen or sharply upcurved. In *P. ultimum* usually a single antheridium is present, while *P. debaryanum* may form 2 to 6 per oogonium. The oospore wall is thicker for *P. ultimum* (2 u), otherwise it corresponds pretty well with the following description given by Middleton (73) for *P. debaryanum:*

"Hyphae branched, usually 5 u in diameter, septate in old cultures. Sporangia spherical or oval, terminal or intercalary, 15 to 26 u, average 19 u, in diameter, germinating either by germtubes or zoospores. Oogonia smooth, terminal or intercalary, usually spherical, 15 to 28 u, average 21 u, in diameter. Antheridia 1 to 6 per oogonium, monoclinous and diclinous, when monoclinous arising some distance below the oogonium, not adjacent to it. Oospores smooth, aplerotic, 12 to 20 u, average 17 u, in diameter, germinating by means of germ tubes."

Epidemiology: Particularly troublesome in cool soil in the spring. The experimental data of Middleton (73) show slight growth at 1°C. within 24 hours, maximum growth about 28°C. and no growth above 37°C. Both species are similar in temperature requirements.

Control: Use plump fresh seed and avoid hollows where water will be retained. Applications of ammonium phosphate at the rate of 100 lbs. per acre have been suggested by Sprague (104) for control on wheat. Red copper oxide was found by Horsfall (56) to provide control when used as a seed treatment or as a spray (1 oz. to 3 gal. water) on the seedlings. Zerbam (Zerlate), thiuram (Arasan), Spergon, and zineb (Parzate, Dithane) have been used successfully on the Rhode Island plots.

The comprehensive monograph of the genus Pythium by Middleton (73) gives reports on the species listed below as causing disease on the hosts named:

Pythium aristosporum Vanterpool on *Agropyron trachycaulum* (38).

Pythium graminicolum Subrm. on *Agropyron cristatum, A. inerme, A. intermedium, A. michnoi, A. repens, A. smithii, A. trichophorum, Festuca idahoensis* (15, 106).

Pythium intermedium de Bary isolated from *Agrostis stolonifera, Festuca duriuscula,* and *Lolium perenne* var. *westerwoldicum* (117).

Pythium irregulare Buisman isolated from *Agrostis stolonifera, Festuca duriuscula, Lolium perenne* var. *westerwoldicum* (116, 117); *Agropyron cristatum, Festuca elatior* (38).

(Plate 1)

Courtesy Mallinckrodt Chemical Works

Top, Brown patch showing typical smoke ring.
Bottom, Brown patch closeup of wilting at margin.

(Plate 2)

Courtesy Mallinckrodt Chemical Works

Top, Dollar spot injury of creeping bent.
Bottom, Dollar spot mycelium killing turf.

(Plate 3)

Courtesy Mallinckrodt Chemical Works

Top, Snow scald injury as snow melts.
Bottom, Snow scald fungus on Seaside bent.

(Plate 4)

Courtesy Mallinckrodt Chemical Works

Top, Mushroom fairy ring of *Marasmius oreades*.
Bottom, Copper spot damage of velvet bent.

Pythium mamillatum Meurs. isolated from *Agrostis stolonifera* and *Festuca duriuscula* by Van Luijk (116, 117).

Pythium periilum Drechsler on *Agropyron repens* (38).

Pythium torulosum Coker & Patterson on *Agrostis stolonifera* and *Festuca duriuscula* by Van Luijk (116).

Pythium vexans de Bary cultured from *Agropyron inerme, A. smithii, Festuca ovina, Poa nevadensis* and *P. palustris* (101).

Pythium volutum Vanterpool & Truscott isolated from *Agrostis stolonifera, Festuca duriuscula, Lolium perenne* var. *westerwoldicum* by Van Luijk (116, 117).

ROOT ROT

Symptoms: Seedlings at the first and second leaf stage yellow and die in small areas, then under favorable conditions the disease may spread until reseeding is necessary. Diseased roots and leaf sheaths contain numerous, microscopic spherical or oval cysts, 15 to 45 by 12 to 30 u.

Distribution: General.

Hosts: Agropyron repens, Agrostis spp.

Pathogen: Cladochytrium graminis Busgen; possibly *C. caespitis* Griff. & Maubl. (17, 59).

Mycelium intracellular, irregular, aseptate, contents densely granular, arises as short branches from a small spherical body. Sporangia develop as vesicles formed from hyphal swellings. Resting sporangium ellipsoidal or spherical, pale yellow, wall smooth on the outside, 3 to 5 u thick. Zoospores unicilliate.

Epidemiology: Occurs during unusually wet conditions; spread checked by dry weather.

Control: Little investigated; Jones (59) used Cheshunt mixture successfully. Chemicals effective against damping off are suggested.

ROOT SCORCH

Symptoms: This disease may attack seedlings or appear the second season after seeding as more or less extensive brown patches where grass has a withered, burned appearance. Stellate cysts develop in roots and root hairs.

Distribution: Cosmopolitan.

Hosts: Agrostis spp., *Festuca* spp., *Lolium perenne.*

Pathogens:

1. *Olpidium brassicae* (Wor.) Dang.

Mycelium absent. Zoosporangia solitary or aggregated in the host cell, thin-walled, spherical (12 to 20 u in diameter) to elongate (25 to 220 by 20 to 45 u), opening by 1 to 4 exit tubes which vary in length according to the distance to the host surface. Zoospores uniciliate, 3 u in diameter. Resting sporangia (cysts) usually spherical, 8 to 25 u in diameter, or if oval up to 30 u in length; endospore thin, smooth, free from exospore; exospore wrinkled with ridges up to 3.5 u in height, stellate in form with 6 to 9 points in optical section (39, 92).

2. *Olpidium agrostidis* Sampson.

Mycelium absent. Zoosporangia 30 to 68 by 10 to 23 u, 1 to 4 short discharge tubes. Zoospores 1-ciliate, hyaline, subglobose, about 2.2 u in diameter. Resting spores solitary or aggregated, elliptical or ovoid, smooth, hyaline, thick-walled, 20 to 30 by 9 to 17 u in diameter (91, 93).

RUSTS

This group of diseases is relatively unimportant on mown turf, but may be quite serious in the production of a seed crop.

1. Black Rust

Symptoms: Elongate reddish or black pustules largely confined to the stem and leaf sheath, but on glumes and leaves when severe.

Distribution: Cosmopolitan.

Hosts: Agropyron spp., *Agrostis* spp., *Festuca* spp., *Lolium* spp., *Poa* spp.; alternate hosts *Berberis* spp., *Mahonia* spp.

Pathogen: Puccinia graminis Pers. var. *agrostis* Erikss., var. *poae* Erikss. & Henn.

Uredia caulicolous and epiphyllous, reddish to reddish-brown; urediospores oblong or ellipsoid, 13 to 24 by 21 to 42 u, wall golden brown at maturity, 1.5 to 2.0 u thick, strongly echinulate, the pores 4, rarely 5, equatorial. Telia chiefly caulicolous, blackish-brown; teliospores ellipsoid or oblong-clavate, 16 to 23 by 35 to 58 u, rounded or narrowed both above and below, moderately constricted at septum; wall dark chestnut-brown above, paler below, 1 to 1.5 u thick at sides, 5 to 10 u above; pedicel colored next to spore, as long as spore or longer (6).

Epidemiology: Many physiological races known and designated by number.

Control: Removal of susceptible species of *Berberis* or *Mahonia* from the vicinity. If damage is serious, dusting with sulfur or ferbam may prove worthwhile.

2. Crown Rust

Symptoms: Defoliation. Bright orange pustules (urediosori) form raised swellings on both leaf surfaces. Later, black, linear, at first covered, then rupturing lesions (teliosori) appear on the lower leaf surface.

Distribution: Cosmopolitan.

Hosts: Agropyron spp., *Agrostis* spp., *Festuca elatior*, *Lolium* spp., *Poa* spp.; alternate hosts, *Rhamnus* spp., *Berchemia scandens*, *Elaeagnus commutata*, *Shepherdia canadensis*.

Pathogen: Puccinia coronata Corda var. *agropyri* Erikss., var. *agrostis* Erikss., var. *festucae* Erikss., var. *lolii* Erikss.

Uredia amphigenous, brownish-yellow, sometimes with few colorless paraphyses; urediospores globoid or broadly ellipsoid, 16 to 20 by 18 to 24 u; wall pale yellow, 1 to 1.5 u thick, finely echinulate, the pores 6 to 8, scattered, indistinct. Telia amphigenous, long covered by the epidermis, occasionally with few subepidermal paraphyses; teliospores clavate-oblong, 13 to 19 by 30 to 67 u, with 3 to 10 digitate projections above, narrowed below, slightly or not constricted at septum; wall chestnut-brown above, paler

below, 1 to 1.5 u thick at sides, 7 to 15 u above, including projections; pedicel tinted, very short (6).

Epidemiology: Urediospores survive the winter and serve as inoculum.
Control: Remove the alternate hosts from the vicinity, if practical.

3. Leaf Rust

Symptoms: Oblong, scattered, 0.5 to 1 mm., reddish to cinnamon-brown pustules on the upper leaf surface and later blackish brown, broadly oblong, 0.5 to 1 mm. pustules on the lower leaf surface.

Distribution: Cosmopolitan.

Hosts: Many grass plants, as the pathogen is a complex species of many varieties.

Pathogen: Puccinia rubigo-vera (DC.) Wint. var. *agropyri* (Erikss.) Arth., var. *agropyrina* (Erikss.) Arth., var. *apocrypta* (Ellis & Tr.) Arth., var. *impatientis* (Arth.) Mains.

Uredia usually amphigenous, cinnamon-brown, with few or no colorless paraphyses; urediospores globoid or broadly ellipsoid, 13 to 24 by 16 to 32 u; wall pale cinnamon-brown or yellowish, 1 to 2 u thick, finely echinulate, the pores 6 to 8, sometimes 4 to 6, scattered, usually distinct. Telia chiefly hypophyllous, oblong, scattered, long covered by epidermis, with thin layers of dark-brown subepidermal paraphyses; teliospores oblong clavate to cylindric, 13 to 24 by 32 to 65 u, or on some hosts 10 to 18 by 26 to 45 u, rounded or truncate above, narrowed below, usually not constricted at septum; wall chestnut-brown, paler below, 1 u or less thick at sides, 3 to 7 u above; pedicel colored, very short (6).

Epidemiology: Brown (14) noted marked variation in reaction to the rust by plants of the same species of grass inoculated with the same culture under identical conditions. He believes this probably is due to genetic impurity.

Control: If feasible, and damage is severe, remove *Anemone, Aquilegia, Clematis, Ranunculus, Impatiens, Thalictrum* and similar alternate hosts.

Some other rusts on the grasses under discussion are:

1. *Puccinia cockerelliana* Bethel.
2. *Puccinia crandallii* Pam. & Hume.
3. *Puccinia cynodontis* Lacroix.
4. *Puccinia festucae* (DC.) Plowr.
5. *Puccinia jacksonii* A. & F.
6. *Puccinia liatridis* (Webber) Bethel.
7. *Puccinia montanensis* Ellis. Brown Stripe Rust.

Hosts: Agropyron inerme, A. trachycaulum, A. spicatum, A. subsecundum, Lolium multiflorum.

8. *Puccinia pattersoniana* Arth.

Hosts: Agropyron dasytachyum, A. inerme, A. spicatum, A. subsecundum.

9. *Puccinia piperi* Ricker.
10. *Puccinia poae-sudeticae* (Westend.) Jorstad. Bluegrass Leaf Rust.

Hosts: Poa ampla, P. compressa, P. nervosa, P. pratensis, P. scabrella, P. vaseyochloa.

11. *Puccinia poarum* Niels.

12. *Uromyces dactylidis* Otth.

Host: Poa canbyi.

SMUTS

1. Bluegrass Blister Smut

Symptoms: Gray-black, blister-like areas develop in the leaves due to subepidermal masses of chlamydospores. A fine, dry series of dot-like masses of conidia (sporidia) appear scattered in rows along the surface of the blisters.

Distribution: Oregon, Washington, Minnesota, N. Dakota.

Hosts: Poa annua, P. compressa, P. juncifolia, P. pratensis, P. vaseyochloa.

Pathogens: Entyloma spp.; *E. irregulare* Johans., *E. crastophilum* Sacc., *E. bingenensis* Zundel.

E. irregulare — sori in leaves, subcircular to oblong, about 1 mm., distinct or somewhat merging into one another, lead color, long covered by epidermis. Chlamydospores almost colorless to very light brown, very irregular to subspherical or polyhedral, chiefly 8 to 14 u, most elongated, occasionally 17 u in length. Conidia often present in very small white clusters on sori (16). Differs from *E. crastophilum* by the lighter colored, more irregular spores and the possession of conidia.

2. Stripe Smut

Symptoms: Long, dark, narrow striations or stripes develop in the leaves, and as the sori mature spores are freed and the blade splits into ribbons. Systemic infected plants make poor vegetative growth, while inflorescences are absent or stunted.

Distribution: General in U.S.A. and most of Europe, Australia.

Hosts: Agropyron cristatum, A. inerme, A. repens, A. semicostatum, A. smithii, A. spicatum, A. subsecundum, A. trachycaulum, Agrostis alba, A. exarata, A. humilis, A. palustris, A. perennans, A. rossae, A. scabra, A. tenuis, Festuca idahoensis, F. obtusa, F. ovina, F. ovina var. *brachyphylla, Lolium perenne, Poa alpina, P. annua, P. compressa, P. juncifolia, P. languida, P. longifolia, P. pratensis, P. reflexa* (35).

Pathogen: Ustilago striiformis (Westend.) Niessl.

A collective species with forms differing in host specialization, spore size, and markings of the epispore. Physiologic form names proposed by Davis (25). F_1 hybrids by crossing *U. striaeformis* X *U. bullata* on *Agropyron trachycaulum* have been obtained by G. W. Fischer (36). Chlamydospores brown, spherical or slightly oval; covered with short spines (under 1 u in length) which appear as a fine irregular network; 8 to 12 u, sometimes 13 to 17 u.

Epidemiology: Infection occurs in seedlings or young bluegrass tillers, presumably from spores in the soil, or in the seed (35). Perennial mycelium may overwinter in the host. Mature spores germinate in water at tempera-

tures of 7°C. minimum, 22°C. optimum, and 35°C. maximum. Drying decreases viability of mature spores.

A controversy was started by Davis (23) who reported that spores required a maturing period of 240 to 265 days before germination. Kreitlow (62) germinated chlamydospores from some *Agrostis alba* plants at room temperature in tap and distilled water without "after-ripening" when uncovered and exposed to atmospheric conditions. He also found (63) that spores removed from diseased plants growing at 32°C. would germinate 90 percent immediately without after-ripening, while maturation was required of spores taken from plants growing at 1.5°C., 10°C., and 20° to 25°C. Kreitlow (64) then reported that spores could be "after-ripened" within 10 to 25 days while in contact with water by proper temperature manipulation. *Ustilago striiformis* chlamydospores from bluegrass (*Poa pratensis*) start to germinate after 5 days in horse or cow dung infusion and are 60 to 90 percent germinated within 25 days. The stimulatory factor is thermostable. If spores are soaked 15 hours in concentrated dung infusion and then dispersed in distilled water, germination began within 5 hours and reached 60 to 90 percent in 20 hours. The spores can be soaked for at least 5 months in a concentrated dung infusion at 25° or 2°C. without loss of viability and will germinate promptly on dispersion in distilled water. The fungus may be grown on potato dextrose and other agar media (35, 66, 63) with critical temperatures about 5°C. minimum, 20° to 25°C. optimum, and 35°C. maximum.

Control: Stripe smut resistance must be considered in any bluegrass improvement program, as a survey by Wellhausen, et al. (120) in West Virginia and Pennsylvania showed the disease to be widespread and in up to 25 percent of the plants examined. Susceptible clones of *P. pratensis* can be eliminated from resistant ones by a method of artificial inoculation devised by Leach, et al. (66).

3. Bunt

Symptoms: Seed filled with black spores; plants stunted.

Distribution: Oregon, Rhode Island. Serious in seed-growing, causing up to 80 percent infected, nonviable seed.

Hosts: Agrostis canina, A. palustris.

Pathogen: Tilletia pallida G. W. Fischer.

Sori in the ovaries, 0.5 to 1 mm. in length; sterile cells few, smaller than the spores, hyaline, thin-walled, smooth; spore mass light brown or buff; spores pale yellowish-brown to hyaline, globose, with 3 u deep, hyaline, irregular reticulations, often appearing cerebriform or even coarsely verrucose, 16 to 24 u, mostly 19 to 20 u in diameter (34).

Another bunt caused by *Tilletia decipiens* (Pers.) Koern. has been reported on *Agrostis stolonifera* and *A. tenuis* in Germany, Denmark and Sweden. The chlamydospores are brown, ridges on epispore form a relatively deep irregular mesh; 23 to 26 u.

Other, less prevalent, smuts reported (126, 98, 34) are:

1. Flag Smut—*Urocystis agropyri* (*Preuss*) Schrot.

Hosts: Agropyron inerme, A. smithii, A. subsecundum, A. trachycaulum, Agrostis alba, Poa ampla, P. canbyi, P. nervosa, P. pratensis, P. secunda.

2. Fescue Smut— *Ustilago mulfordiana* Ell. & Ev. on *Festuca octoflora, F. reflexa.*

3. Head Smut—*Ustilago bullata* grows in culture at optimum of 20° to 25°C.

Hosts: Agropyron dasystachyum, A. trachycaulum.

POWDERY MILDEW

Symptoms: Foliage appears yellow or chlorotic, gray-white weft of mildew (mycelium, conidiophores, conidia) appears chiefly on upper leaf surfaces. Sometime in late summer small brownish-black dots (perithecial fruit-bodies) appear among the mycelium. Seed from infected plants is small and shrivelled.

Distribution: Cosmopolitan, worldwide.

Hosts: Agropyron repens, A. spicatum, A. trachycaulum, A. trichophorum, Festuca idahoensis, F. occidentalis, F. octoflora, F. rubra, F. rubra var. *commutata, F. thurberi, Poa ampla, P. annua, P. arachnifera, P. canbyi, P. compressa, P. curta, P. glauca, P. juncifolia, P. leptocoma, P. mutalensis, P. nemoralis, P. palustris, P. pratensis, P. scabrella, P. secunda, P. trivialis, P. vaseyochloa.*

The degree of susceptible or resistant reaction may depend on variation within the grass species, but chiefly on pathogenic specialization (physiologic races) of the pathogen.

Pathogen: Erysiphe graminis DC.

Mycelium composed of septate hyphae on the outside of the epidermal cells; appendaged (branched) haustoria. Conidia formed in chains on short upright conidiophores, each having a slightly swollen base; spores hyaline, oval, 25 to 33 by 14 to 17 u. Perithecia dark, spherical, 135 to 280 u in diameter, scattered or in clusters; appendages simple, poorly developed; contain several pedicillate asci, 70 to 108 by 25 to 40 u. Ascospores ovoid, 20 to 23 by 10 to 13 u, 8 per ascus.

Control: If serious, sulfur dusts or wettable sulfur sprays will reduce damage. Potash fertilization has a deterrent effect (69). Striking differences in susceptibility are exhibited by clones of grasses.

BLUEGRASS EYESPOT

Symptoms: Lesions light-brown with a distinctive straw-color center and a surrounding border or halo of yellow; at first very small, circular, later enlarging to nearly the width of the leaf or elongating longitudinally on the leaf for some distance; spots finally becoming dull brown and eventually fading to straw color (102).

Distribution: Western Oregon.

Host: Poa pratensis.

Pathogen: Cercosporella poagena Sprague.

Conidiophores hyaline, obscure, arising from hyaline thinly formed aggregating hyphae; conidia hyaline, elongate, broadly filiform to obclavulate, bases blunt, tapering, apices either abruptly acuminate or attenuate at the tips, 4- to 7-septate (mostly 4-septate), 45 to 75 — [90] by 3.6 to 4.6 — [5] u.

TAR SPOT (Black Leaf Spot)

Symptoms: Spots elongated, grayish-violet to dark olive-green, becoming black, on both surfaces of living leaves; leaves yellow and wither prematurely.

1. *Pathogen: Phyllachora graminis* (Fr.) Fuckel.

Conidia (*Hadrotrichum virescens* Sacc. & Roum.) single, terminal, subspherical to spherical, echinulate, brown-olivaceous, non-septate, [7] — 11 to 13.5 — [15] u in diameter; conidiophores unbranched, cylindrical, closely packed, septate only at the base, brown-olivaceous, [25] — 29 to 32 — [44] by 6 to 8 u (46, 93). Perithecia single or multiple loculi in a subepidermal stroma, usually on upper leaf surface, at first depressed, later erumpent (46). Clypei amphigenous, scattered, long elliptical to linear, often fusiform in outline, 0.1 to 1.0 mm. wide by 0.2 to 5.0 mm. long, frequently confluent, black; fructification compound with many ascocarps; asci cylindrical, 8 to 10 by 70 to 100 u; 8 ascospores, ellipsoid, 4.5 to 6 by 9 to 12 u, uniseriate (83).

Distribution: Worldwide, chiefly in north temperate zone.

Hosts: Agropyron cristatum, A. repens, A. trachycaulum, Agrostis alba, A. stolonifera, A. tenuis, Festuca elatior, Lolium perenne.

2. *Pathogen: Phyllachora sylvatica* Sacc. & Speg.

Clypei mostly hypophyllous, scattered or often gregarious, roundish or oval in outline, 0.4 to 0.8 mm. wide by 0.5 to 1 mm. long, black; fructification compound with few ascocarps; asci cylindrical or narrowly elliptical, 10 to 15 by 75 to 100 u; 8 ascospores, ellipsoid to fusiform or ovoid, 6.0 to 7.5 by 12 to 16 u, biseriate or uniseriate (83).

Distribution: Northwestern U.S.A., northern Italy.

Hosts: Festuca dertonensis, F. idahoensis, F. megalura, F. occidentalis, F. ovina var. *duriuscula, F. rubra.*

CHAR SPOT

Symptoms: Leaf lesions tawny with yellow margins, circular becoming typically elliptical to elongate, pointed at each end, covered with dull black to creosote-brown stromatic fungus tissue. Pycnidia sometimes separate as small black dots at lesion margin, otherwise obscured by stroma.

Distribution: U.S.A., Europe.

Hosts: Agropyron spicatum, A. trachycaulum, Agrostis alba, A. hallii, A. oregonensis, A. scabra.

Pathogen: Septogloeum oxysporum (Sacc.) Bommer & Rousseau.

Pycnidia subglobose, brown, embedded in stroma or separate at margin of lesions, 80 to 160 u in diameter, with ostioles up to 60 u in diameter. Conidia yellow to subhyaline, fusoid, often flattened slightly on one side, subtruncate at the base, tapering to an obtusely pointed apex; 0- to 3-septate, mostly 2-septate; 21 to 31 by 3.2 to 4.4 u on *Agrostis* sp. Conidiophores hyaline, globular to cuspidate in grayish masses. Perithecia

in stroma, globose, up to 250 u in diameter; walls creosote-brown, of compacted polygonal cells. Close to *Dothidella aristidae,* but life history study is needed according to Sprague (104).

Epidemiology: Epidemic on grasses in humid areas.

SEPTORIA LEAF SPOTS

1. *Symptoms:* Brown or fuscous leaf blotch.

Distribution: Northern U.S.A., Asia, Europe.

Hosts: Agropyron spp.

Pathogen: Septoria agropyrina Lobik.

Mycelium cottony buff. Pycnidia large, brown, ostiolate. Conidia hyaline, 3-septate, obclavate to subcylindric, apically pointed, [30] — 40 to 60 — [65] by 2.5 to 4.5 — [4.5] u.

2. *Symptoms:* Lesions gray to straw-color, scattered on the tips of blades; pycnidia obscure, scattered.

Distribution: Oregon, Wyoming, Alaska, probably worldwide.

Hosts: Agrostis diegoensis, A. exarata, A. palustris, A. rossae, A. scabra.

Pathogen: Septoria calamagrostidis (Lib.) Sacc.

Pycnidia characteristically strongly flattened, subepidermal, dark brown, appearing black in leaf spots, 50 to 140 by 50 to 180 u in diameter and 50 to 85 u deep; wall thickness varies with time and collection. Conidiophores small, blunt, bottle or awl-shape, 2 to 5 u long. Conidial mass slimy, usually darkening to coffee-brown. Conidia filiform, curved, often sinuous, distinctly 0- to 5-septate, mostly 3-septate, [25] — 40 to 65 — [71] by [0.8] — 1 to 1.1 — [2.1] u (103).

Epidemiology: Appears in late winter or early spring in Oregon particularly on Seaside creeping bent.

3. *Symptoms:* A speckled leaf blotch due to pale gray, tan or fuscous lesions; numerous pycnidia, imparting a salt and pepper aspect.

Distribution: Northern and northwestern U.S.A., China.

Hosts: Agropyron dasystachyum, A. inerme, A. repens, A. smithii, A. spicatum, A. subsecundum, A. trachycaulum.

Pathogen: Septoria elymi Ellis and Everhart.

Pycnidia flattened, ostiolate, golden-brown or brown, subepidermal, thin-walled (5 to 13 u), ellipsoid, [65]— 95 to 130 — [200] by 60 to 115 — [160] u. Conidiophores usually subulate, [3] — 4.8 to 4.5 — [7.5] by [0.9] — 1.5 to 1.6 — [2.9] u, sometimes 1-septate, 3 to 6 u long. Conidia clear hyaline, filiform-bacillar or filiform-cylindric, blunt at the base and narrowed at the apex, 0- to 3-septate, mostly 2- or 3-septate, [15] — 30 to 25 — [42] by [1.2] — 1.6 to 2 — [2.3] u. Cultures develop as small pale cream, later flesh pink colonies of yeasty conidial masses; becoming covered with mouse gray, short, felty growth (103).

4. *Symptoms:* Fuscous to deep chocolate-brown leaf spot paler in center, surrounded by lighter areas.

Distribution: California, France.

Host: Lolium multiflorum.

Pathogen: Septoria loligena Sprague.

Pycnidia light golden-brown to brown, thin-walled, subglobose, not prominent, 80 to 120 u in diameter. Conidia clear hyaline, bacillar, 0- to 3-septate, [25] — 28 to 45 —

[50] by 2.7 to 4.1 u. Cultures are slow growing, cottony, rose-buff color, with irregular or fluted margin.

5. *Symptoms:* Lesions greenish mottled, later gray, straw-colored or brown blotches, finally bleached, off-white, variable in shape; pycnidia present.

Distribution: Prevalent in northern U.S.A., Canada, Europe, Morocco, China.

Hosts: Poa ampla, P. annua, P. arida, P. canbyi, P. compressa, P. cusickii, P. gracillima, P. howellii, P. kelloggii, P. nervosa, P. nevadensis, P. pratensis, P. scabrella, P. secunda, P. vaseyochloa.

Pathogen: Septoria macropoda Passerini.

A polymorphic species with 2 varieties and a number of physiologic races (103). Pycnidia irregular, subepidermal, slightly erumpent, mostly flattened and appressed to the upper leaf epidermis, some subglobose, golden-brown to brown, ellipsoid, [60]—65 to 135—[190] by [66] — 70 to 160 — [240] u and [40] — 50 to 80 — [90] u high; walls thin (5 to 11 u), light brown, consist of 3 to 4 layers of oblong to irregularly polyhedral cells merging into 2 to 4 inner layers of hyaline cells; conidiophores subulate or subcylindric (but vary with host species), 2 to 3 or 4 to 7 by 1.5 to 2.5 u, papillate. Conidia filiform to narrowly filiform-clavate, 1- to 3-septate, [30] — 40 to 40 — [75] by [1.] — 1.3 to 1.7 — [2.4] u. Cultures slow-growing, mucose, later carbonaceous, sometimes cream-colored, tawny, isabelline to pale flesh conidial exudate with gray overtones.

Epidemiology: Appears during foggy or humid weather.

6. *Symptoms:* Blotch of glumes.

Distribution: Northern U.S.A., Canada, Europe.

Hosts: Agropyron repens, A. smithii, A. trachycaulum, Poa palustris, P. pratensis.

Pathogen: Septoria nodorum Berkeley. Possibly a *Stagonospora* (103, 104).

Mycelium cottony, buff or light-colored. Conidia bacillar, cylindric to subfusoid, typically rounded at both ends and slightly to distinctly constricted at the septa, generally 2- or 3-septate, sometimes 0- or 1-septate, [15] — 25 to 30 — [45] by [2.3] — 2.5 to 4.3 u.

7. *Symptoms:* Grass assumes a yellowish-brown cast. Small dark brown or light purple, finally straw-colored spots, first on leaf sheaths spreading to all blades on the shoot; leaves yellow at tips. Plants defoliated but rarely killed; pycnidia pale, obscure.

Distribution: Northern U.S.A.

Hosts: Poa ampla, P. arachnifera, P. autumnalis, P. canbyi, P. compressa, P. juncifolia, P. nevadensis, P. palustris, P. pratensis, P. secunda.

Pathogen: Septoria oudemansii Saccardo.

Pycnidia yellow-brown, subglobose, somewhat erumpent, [85] — 100 to 180 u in diameter; wall consists of a yellow-brown outer layer, 6 to 7 u thick, surrounding a hyaline layer 4 to 4.5 u thick; conidiophores bulbous, 4 to 5 by 1.8 to 2.2 u. Conidia (macrospores Ascochyta-like), hyaline, typically cylindrical but slightly thicker at the central septum, sometimes tapering to lanceolate, contents bright hyaline with small uniform oil drops adjacent to the septum, 1- to 3-septate, sometimes non-septate, [12] — 13 to 19 — [24] by [1.4] — 1.7 to 1.8 — [2.8] u; microspores range from 4 by 2 u up to macrospore size. Cultures form white branching, cottony mycelium, later margin turns olive-gray with olivaceous substratum, finally become mottled gray, cream, white, and dull buff intermingled. Pycnidia formed in abundance at 25°C. on sterilized bluegrass leaves (42, 103).

Epidemiology: Appears in cool, wet seasons, particularly in the spring.

8. *Symptoms:* White leaf spot becoming straw-colored, pycnidia scattered.
Distribution: Northern and northwestern U.S.A.
Hosts: Agrostis hallii, A. scabra.
Pathogen: Septoria secalis Prill. & Del. var. *stipae* Sprague.

Pycnidia scattered, globose, 130 to 190 u in diameter, ostiole erumpent; wall thin. Conidia (macrospores) hyaline, bacillar, 1- to 3-septate, typically 3-septate, [37] — 42 to 54 — [62] by [2.6] — 2.8 to 3.2 — [3.4] u; (microspores) *Phoma*-like 5 to 10 by 0.5 to 0.7 u. Cultures slow growing, white, fragile, finally forming circular, pale buff compact mat 1 inch across (103).

9. *Symptoms:* Small, vague, greasy brown spots on living leaves; sometimes straw-colored, indefinite blotches.
Distribution: Widespread in northern plains and northwest U.S.A.
Hosts: Festuca dertonensis, F. elatior, F. idahoensis, F. octoflora, F. ovina, F. rubra, F. rubra var. *commutata.*
Pathogen: Septoria tenella Cooke & Ellis

Pycnidia creosote-brown, more or less elliptical in cross section, [60] — 80 to 120 by 95 to 130 u; wall of tightly crushed, oblong to polyhedral cells, 0.5 to 3 u wide; conidiophores cylindrical, very narrowly subulate, about 1 u wide and 4 u long. Conidia variable, commonly filiform, somewhat curved, 0- to 2-septate, [5] — 44 to 45 — [70] by [0.8] — 1.6 to 1.5 — [2.1] u. Cultures produce cream-white, soon flesh-pink eventually slimy cocoa-brown masses of conidia; later become carbonized by development of stromatic hyphae.

10. *Symptoms:* Lesions abundant, gray, later fuscous to brown (cinereous), indeterminate, sublinear to irregular, or on leaf tips; pycnidia obscure.
Distribution: Northwestern U.S.A.
Hosts: Agrostis alba, A. castellana, A. exarata, A. tenuis.
Pathogen: Septoria triseti, Speg. emend Sprague.

Pycnidia black-brown, ostiolate, substomatal, globose to flattened at the ostiolar end where it is appressed against the leaf epidermis, [40] — 50 to 80 — [100] u in diameter; wall formed of irregular, oblong-polyhedral, parenchymatous cells, in strands, which converge in radii towards the ostiole, brown outer cells 2 layers thick (3 u) with 2 or 3 inner hyaline layers (3 to 5 u thick); conidiophores narrowly bulbous-cylindric, 2.5 to 3.5 u. Conidia filiform to subbacillar or sometimes narrowly fusiform, straight, bent or less often moderately curved, 0- to 1-septate, [16] — 18 to 35 — [43] by [0.8] — 1.3 to 1.7 — [2.0] u.

11. *Symptoms:* Lesions indefinite, first appear green, later yellowish, mottled, or blotched, becoming fuscous to deep brown; pycnidia moderately prominent, scattered or in rows parallel to leaf veins.
Distribution: North America, Europe.
Hosts: Lolium multiflorum, L. perenne.
Pathogen: Septoria tritici Roberge var. *lolicola* Sprague & A. G. Johnson.

Pycnidia subglobose to globose, sometimes ellipsoid, [80] — 120 to 150 by [90] — 120 to 150— [180] u, pale to deep amber; ostiole 10 to 25 by 10 to 30 u in diameter; wall relatively thin (15u), usually 5 to 6 cells thick, 1 or 2 outer layers of oblong cells, 2 or 3 inner layers of intertwined lighter polyhedral cells which give rise to hyaline cells producing slightly pyriform to subcylindric conidiophores measuring 4 to 8 u long and 1.5 to 2.3 u wide. Conidia hyaline, sometimes with coarse protoplasm, sometimes containing small oil drops, slightly enlarged at base, curved or sinuous, distal cells tapering to whip-like, 0- to 5-septate, commonly 1- to 3-septate, 21 to 85 by 1.3 to 2.8 u (103).

ASCOCHYTA LEAF SPOTS

Symptoms: Spots vague or variable in shape, usually pycnidia occur in straw-colored, bleached areas. Conidia are diagnostic.

Pathogens:

1. *Ascochyta agropyrina* (Fairman) Trotter.

Distribution: Northwestern U.S.A.

Hosts: Agropyron bakeri, A. caninum, A. cristatum, A. michnoi, A. semicostatum, A. sibiricum, A. smithii, A. spicatum, A. subsecundum, A. trachycaulum, A. trichophorum.

2. *Ascochyta desmazieri* Cav. causes well defined chocolate brown spots.

Distribution: California, Oregon, Washington.

Hosts: Lolium multiflorum, L. perenne.

Pathogen: Spores cylindrical, 12 to 20 by 2.3 to 3.1 u.

3. *Ascochyta elymi* Tehon and Daniels.

Host: Agropyron trachycaulum.

4. *Ascochyta graminea* (Sacc.) Sprague & Johnson.

Distribution: South Carolina, North Dakota, California; Italy.

Host: Cynodon dactylon.

5. *Ascochyta hordei* Hara.

Host: Festuca elatior.

6. *Ascochyta sorghi* Sacc. (*A. graminicola* Sacc.). Few red-bordered or fawn-colored lesions on the lower leaves or on the tips of foliage, 5 to 8 mm. broad.

Distribution: North America, Europe.

Hosts: Agropyron smithii, Agrostis alba, Festuca ovina, F. rubra, Lolium sp., *Poa gracillima, P. pratensis.*

Pathogen: Pycnidia gregarious, pale-brown, pallid, lens-shaped, punctiform, open by means of a pore, 100 to 120 u in diameter. Conidia hyaline, subfusoid to fusoid, straight, 1-septate, [10]-14 to 15-[17] by 3 to 4 u.

Epidemiology: Appears in late summer months.

7. *Ascochyta utahensis* Sprague.

Distribution: Utah, Wyoming, Colorado.

Hosts: Agropyron inerme, A. trachycaulum.

SELENOPHOMA "FROGEYE" LEAF SPOTS

Symptoms: Frogeye spots on living leaves, often fading to straw-colored tissues with obscure, minute pycnidia scattered in lines. Similar to but separated from *Septoria* by the non-septate, falcate spores.

Distribution: Common in northern North America, Europe, and Asia.

Pathogens:

1. *Selenophoma donacis* (Pass.) Sprague & Johnson (and varieties).

Hosts: Agropyron albicans, A. cristatum, A. dasystachyum, A. griffithsii, A. inerme, A. repens, A. riparium, A. smithii, A. spicatum, A. subsecundum, A. trachycaulum, Festuca idahoensis, Poa alpina, P. ampla, P. arida, P. canbyi, P. compressa, P. cusickii, P. epilis, P. fendleriana, P. glauca, P. gracillima, P. juncifolia, P. nevadensis, P. palustris, P. pratensis, P. secunda.

2. *Selenophoma everhartii* (Sacc. & Syd.) Sprague & Johnson.
Hosts: Agrostis scabra, Festuca ovina var. *brachyphylla.*

3. *Selenophoma obtusa* Sprague & Johnson.
Hosts: Agropyron subsecundum, A. trachycaulum, Poa secunda.

CERCOSPORA LEAF SPOT OF TALL FESCUE

Symptoms: Oval to elongate spots 0.5 to 8 mm. in length with gray centers and purple to purple-brown borders. First occurring on leaf tips, later spreading down the leaf. The spots coalesce, inciting tip necrosis.

Distribution: Kentucky, Texas.

Host: Festuca arundinacea.

Pathogen: Cercospora festucae Hardison.

Conidiophores are pale to medium olivaceous-brown near the base, paler and sometimes tapering slightly toward the tip, 3 to 5 by 39 to 800 (mean 355) u long, 0- to 18-septate, longer conidiophores 1 to 2 geniculate, almost straight, with rounded truncate tip, typically not branched, occasionally branching, arising singly or in groups of 2 to 32 divergent stalks, usually from stomata, occasionally from small connecting cells, stromata none or a few brown cells. Conidia are hyaline, acicular, curved when mature, 4- to 32-septate, base truncate tapering to a rounded tip 1.6 to 4.9 by 40 to 361.5 (mean 215) u long, germinating from any cell.

Epidemiology: Appears on vigorous new leaves in April, progresses through the summer and subsides in October.

WHITE HEADS

Symptoms: Seed heads or panicles wither before becoming fully expanded, later appear silvery and eventually become blanched. The seeds are aborted. Lesions are usually found just above the uppermost node of the culm and oftentimes during moist weather copious mycelium grows from decayed areas of the culm.

Distribution: Maine, Nebraska, Massachusetts, New York, Wisconsin, Rhode Island, Pennsylvania, Canada, Argentina, Italy.

Hosts: Agrostis spp., *Festuca* spp., *Poa* spp.

Pathogen: Fusarium poae (Pk.) Wr.

Mycelium cobweb-like, carmine red or buff; microspores pip- or citron-shape, 0- to 1-septate (*Sporotrichum*), 6 to 14 by 4 to 7 u; macrospores sickle-shape, heel cell not evident, pointed at both ends, 3- to 5-septate, 18 to 36 by 3 to 4.2 u.

Epidemiology: The causal fungus is disseminated by a mite (*Pediculopsis graminum* Reuter), which also serves to inoculate the grass.

Control: Burning over of the dead grass offers a practical method of control.

CHOKE

Symptoms: A cylindrical collar of creamy to grayish-white, later conspicuous golden-yellow to dark orange stroma around the sheaths of the upper leaves.

Distribution: General in U.S.A., Europe.

Hosts: Agropyron inerme, A. repens, A. smithii, A. spicatum, A. trachycaulum, Agrostis alba, A. stolonifera, A. tenuis, Festuca rubra, F. ovina, Poa bulbosa, P. compressa, P. nemoralis, P. pratensis, P. secunda, P. stenantha, P. trivialis.

Pathogen: Epichloe typhina (Fr.) Tulasne.

Stroma composed of closely crowded white hyphae which extend outward radially and bear terminal conidia. Conidia hyaline, unicellular, elliptical, 4 to 5 by 3 u. Perithecia develop as locules in orange stroma with prominent papillate ostioles. Asci contain 8 ascospores which are hyaline, filiform, 2 u in diameter, jointed and multiseptate.

Epidemiology: The fungus grows on the surface of the host grass only during the summer months from systemic mycelium living from year to year in the rhizome or other vegetative organs. The pathogen is distributed by vegetative propagation of infected perennial grasses and by mycelium within the seed of some. Conidia and ascospores are produced in quantity from May to July; the latter being forcibly discharged and dispersed by the wind. A single stroma may discharge 2,000 to 10,000 spores per minute in moderately dry air so long as the water supply to the host is maintained. Damage is chiefly to seed production.

BROWN STRIPE

Symptoms: Chocolate-colored spots with grayish centers between the veins of leaves and sheaths. Groups of conidiophores emerge through the stomata in the upper epidermis while the base of the conidiophores appear as small black spots on the lower surface (93).

Distribution: North America, Europe.

Hosts: Agropyron caninum, A. cristatum, A. dasytachyum, A. griffithsii, A. inerme, A. repens, A. riparium, A. smithii, A. spicatum, A. subsecundum, A. trachycaulum, Agrostis alba, A. castellana, A. exarata, A. hallii, A. oregonensis, A. rossae, A. scabra, Festuca elatior, F. rubra, Poa alpina, P. ampla, P. arida, P. canbyi, P. compressa, P. epilis, P. gracillima, P. interior, P. juncifolia, P. longiligula, P. nervosa, P. nevadensis, P. palustris, P. pratensis, P. secunda, P. stenantha, P. trivialis. Rye grasses appear to be resistant.

Pathogen: Passalora (*Scolecotrichum*) *graminis* (Fckl.) v. Hohnel.

Conidiophores fasciculate, olive-brown, unbranched, irregular, or slightly geniculate, 90 to 100 by 6 to 8 u. Conidia elongated with slightly tapering ends, typically 1-septate, 35 to 45 by 8 to 10 u.

ANTHRACNOSE

Symptoms: Black spots due to sporulating pustules produce a scablike effect. Prominent black spines (setae) usually evident on the lesions.

Distribution: General.

Hosts: Agropyron repens, A. sibiricum, A. trachycaulum, Agrostis alba, A. palustris, Digitaria sanguinalis, Festuca megalura, F. octoflora, F. rubra, F. subulata, Lolium multiflorum, L. perenne, Poa canbyi, P. compressa, P. pratensis.

Pathogen: Colletotrichum graminicolum (Ces.) G. W. Wilson.

Acervuli immersed, then erumpent; prominent black setae, 60 to 120 by 6 to 8 u. Conidia hyaline, non-septate, spindle-form, nearly straight, 18 to 26 by 3 to 4 u, borne on short conidiophores 6 to 12 by 1 to 2 u.

OTHER PATHOGENS REPORTED:

1. *Cercosporella herpotrichoides* Fron. Strawbreaker, Foot Rot.
Hosts: Agropyron cristatum, A. inerme, A. riparium, Poa secunda.

2. *Claviceps purpurea* (Fr.) Tul. Ergot.
Hosts: Agropyron spp., *Festuca* spp., *Lolium* spp., *Poa* spp.

3. *Dilophospora alopecuri* (Fr.) Fr. Twist.
Hosts: Agropyron inerme, A. smithii, A. spicatum, A. trachycaulum, Festuca ovina, Poa scabrella, P. secunda.

4. *Fusarium culmorum* (W. G. Smith) Sacc. Root Rot, Scab.
Hosts: Agropyron cristatum, A. desertorum, A. elongatum, A. inerme, A. sibiricum, A. smithii, A. trachycaulum, Agrostis palustris, A. tenuis, Festuca rubra var. *commutata, Lolium multiflorum, L. perenne, Poa annua, P. juncifolia, P. pratensis.*

5. *Gloeosporium bolleyi* Sprague. Root Necrosis.
Hosts: Agropyron albicans, A. cristatum, A. desertorum, A. elongatum, A. griffithsii, A. intermedium, A. michnoi, A. repens, A. riparium, A. semicostatum, A. sibiricum, A. smithii, A. spicatum, A. subsecundum, A. trachycaulum, A. trichophorum, Agrostis alba, A. scabra, Cynodon dactylon, Festuca arundinacea, F. elatior, F. octoflora, F. ovina, F. pacifica, F. rubra, Poa bulbosa, P. canbyi, P. compressa, P. glaucifoli, P. interior, P. nevadensis, P. palustris, P. pratensis, P. secunda.

6. *Hendersonia crastophila* Sacc. Foot Rot.
Hosts: Agropyron cristatum, A. inerme, A. intermedium, A. repens, A. riparium, A. smithii, A. subsecundum, A. trachycaulum, Festuca idahoensis, F. pacifica, F. rubra, Poa ampla, P. bulbosa, P. canbyi, P. interior, P. pratensis, P. secunda.

7. *Hendersonia culmicola* Sacc. Leaf Rot.
Hosts: Agropyron repens, A. semicostatum, A. spicatum, A. trachycaulum, Festuca idahoensis, F. ovina var. *brachyphylla, F. pacifica, F. rubra, Poa canbyi, P. compressa, P. gracillima, P. palustris, P. pratensis, P. secunda.*

8. *Heterosporium avenae* Oud. Leaf Mold.
Hosts: Agropyron cristatum, Poa gracillima, P. nevadensis.

9. *Mycosphaerella tulasnei* (Jancz.) Rothers. Leaf Mold.
Hosts: Agropyron smithii, A. spicatum, Festuca idahoensis, Poa compressa, P. gracillima, P. pratensis.

10. *Phialea temulenta* Prill. & Delacr. Blind Seed of Ryegrass.
Distribution: North America, Europe, New Zealand.
Hosts: Festuca elatior, F. myuros, Lolium multiflorum, L. perenne.

11. *Phleospora graminearum* Sprague & Hardison. Leaf Spot.
Host: Agropyron repens.

12. *Rhynchosporium secalis* (Oud.) J. J. Davis. Leaf Blotch, Scald.
Hosts: Agropyron dasytachyum, A. elmeri, A. repens, A. semicostatum, A. subsecundum, A. trachycaulum, Agrostis alba, Lolium multiflorum, L. perenne.

13. *Spermospora subulata* Sprague (syn. *Cercosporella subulata* Sprague). Leaf Scald, Blast.
Hosts: Agrostis alba, Festuca rubra, F. rubra var. *commutata.*

14. *Synchytrium graminicola* Sprague. Purple Leaf Speckle.
Distribution: Coastal region of Oregon and Washington, U.S.A.
Hosts: Agrostis alba, A. canina, A. exarata, A. hallii, A. palustris, A. tenuis, Festuca rubra var. *commutata, Poa annua, P. juncifolia, P. pratensis, P. secunda.*
Epidemiology: Seldom destructive, but relatively common during open, wet winter weather.

MUSHROOM FAIRY RINGS

Fairy rings centuries old have been reported in Britain (86). Many species of mushrooms have been found growing in lawns and fairways; among them are *Marasmius oreades, Psalliota* (*Agaricus*) *campestris, P. tabularis, Clitocybe gigantea, Tricholoma gambosum, Lepiota morgani, L. procera,* and *L. naucina.* Three typical forms are described below.
Symptoms: Continuous or interrupted bands of darker green turf in which mushrooms appear from time to time when the grass is kept wet. The mycelium begins growth at the center of the ring and being perennial gradually extends outward at the periphery. Outward spread often averages 5 inches,

but can be up to 24 inches annually. The band-like, underground advancing mycelium breaks down organic matter to products which the roots of associated grasses may assimilate and the foliage becomes dark green. Following the zone of stimulated growth there is often one of dying or dead grass due either to temporary exhaustion of nutrients (45), to the fungus secreting toxic substances and its hyphae penetrating the grass roots and consuming the soft parenchymatous cortical tissue (32), or to the formation of a water impervious soil layer by the dense mycelium (96).

Pathogens:

1. *Marasmius oreades.*

Cap 2 to 6 cm. across, convex, becoming plane with a prominent elevation in the center, rather thin, dry and spongy to touch, smooth, slightly striate when moist, shrivelling in dry weather but reviving with moisture; color varies from pale tan to buff, sometimes golden brown, and darkest in the center; margin thin, often wavy, and in old specimens upturned. Flesh somewhat tough, thick at the center of the cap, cream color, pleasant odor, nutty taste, edible. Gills free from the stem, broad, quite distant, rounded toward the stem end; color faintly tawny and cream when dry. Spores white, elliptical with an oblique basal apiculus, 7 to 8 by 5 u. Stem 4 to 10 cm. long by 2 to 4 mm. thick, solid, very tough, equal, cartilaginous, surface covered with a fine down easily rubbed off (45). (See color plate.)

2. *Psalliota campestris* (Linn.) Fr.

Cap 1.5 to 3 inches across, white, silky or with scales, broadly expanded or nearly flat when mature, peels readily; flesh white becoming pinkish when cut, taste mild, pleasant, edible. Gills pink when young, becoming brown and then black with age, close together, free from the stem. Stem smooth, white or whitish, 2 to 3 inches long, with a ring when young, stuffed cylindric. Spores brownish-purple, elliptic, 6 to 7 by 5 to 5.5 u.

3. *Lepiota morgani.*

Cap 2 to 12 inches across, white with scattered brown scales which merge together at the center; soft fleshy, nearly spherical when young, convex or even depressed at the center when mature. Flesh white, turning reddish and then yellowish where wounded, poisonous. Gills white when young, green when mature, broad, close together, free from the stem. Stem slightly bulbous at the base, firm, stuffed, 6 to 8 inches long, whitish tinged with brown, surrounded by a large ring (annulus) that is often movable. Spores green turning yellow, egg-shaped or slightly elliptical, 10 to 13 by 7 to 8 u.

Control: Organic cadmium fungicides appear to have an inhibiting effect on the formation of some mushrooms from observations on the Rhode Island experimental plots. In connection with commercial mushroom production J. W. Sinden (letter) has noted that dolomitic limestone or magnesium salts will check the development of mushroom fruitbodies. Fairy rings were drenched with iron sulfate in New Zealand, but the necessity for repeated applications and large doses made the measure impractical. Grainger (41) reports that mushrooms have not been found on substrata below pH 5.8, but this requires checking for wild species in view of our observations.

BIRD'S-NEST FUNGI

Symptoms: Small cups, funnels, or inverted bells; singly or in groups; bearing a number of small, seed-like or lentil-shaped bodies (peridioles) resembling a tiny nest full of eggs.

Distribution: Cosmopolitan.

Hosts: Believed to be saprophytic on decaying organic matter in turf.

Pathogen: Cyathus spp.

Fruiting body cupulate or broadly inverted bell-shaped, 10 to 15 mm. high by 8 to 15 mm. across, tapering to a narrow base; gray to dirty-brown outside, at first velvety then smooth; top covered with a thin membrane when young; margin often wavy; inside smooth, leaden to brown color. Peridioles grayish to black, glossy lenticular, 0.2 to 0.4 mm. across, attached by a white strand (funiculus). Spores white, smooth, elliptical, [10] — 14 to 18 — [22] by 8 to 10 u. (45).

Epidemiology: Appear from late May until October.

PUFFBALLS

Lycoperdon spp.

Fruiting body globular, pear, top or flask-shaped, opening at the apex with a distinct mouth; [1] — 3 to 12 — [18] cm. in diameter by [2] — 5 to 7 — [18] cm. high; singly or in clusters; exterior white then yellowish or olivaceous, either smooth or covered with symmetrically arranged, tufted warts or scales, or fine granules easily rubbed off, finally drying and becoming papery; interior solid and white when young, gradually becoming ochre-color as the spores form in upper portion and mature, basal portion sterile. Spores colored; echinulate, verrucose or smooth; globose or elliptical; discharge through apical opening in a cloud when sporophore is touched (45, 87).

Scleroderma spp.

Fruiting body whitish or yellowish; subglobose, obovate, or turbinate, sessile or prolonged into a stem-like base; 2 to 15 cm. in diameter; wall consisting of 1 or 2 layers, thick, tough, leathery or corky, dehiscing irregularly or at the apex in a star-like manner; sporogenous tissue (gleba) gray-white, becoming dark brown or blackish, pulverulant; basidia pear-shape to clavate, bearing 2 to 5 shortly pedicillate spores; capillitium rudimentary; spores colored, globose, echinulate, verrucose or reticulate (87).

Calvatia spp.

Fruiting body very large, globose, top-shaped, ovoid or ellipsoid; 3.5 to 20 inches in diameter by 4 to 13 inches high; apical opening for spore discharge absent; outer wall smooth, scaly or divided into fissured sectors, white changing to brown; inner wall separate, thin, breaking at maturity to liberate the spores; basal portion sterile or partly so; tapering root-like base attached to ramifying mycelium. Sessile on ground, solitary or in a large interrupted ring; edible when flesh is white; July to October. Spores at first yellowish, finally olivaceous to purple-brown when dry; becoming tinder-like (45).

Bovista spp.

Fruiting body similar to *Lycoperdon* but without a sterile basal portion; subglobose to ovoid, [2] — 4 to 7 cm. in diameter by [1] — 3 to 5 — [7] cm. high; outer wall whitish or ochraceous, smooth, thin and papery when mature, opening by an apical aperture or an irregularly torn mouth; attached by a short, root-like basal appendage to the ramifying saprophytic mycelium. Spores colored, smooth, pedicillate, globose, oval or elliptical (45).

SLIME MOLDS

Many species of slime molds have been found forming their spore-containing structures on turf. They are not parasitic but may injure the grass temporarily by smothering or shading. The gelatinous plasmodium ingests microorganisms and decaying organic matter for food, then moves up onto the

grass blades for fruiting. Only two of the most commonly reported types found on lawns are described here.

1. *Mucilago spongiosa* (Leyss.) Morgan.

Symptoms: Cream-yellow plasmodium forms large grayish-white structures filled with a very dark spore mass.

Distribution: Worldwide in temperate regions.

Pathogen: Plasmodium white or yellow.

Aethalia composed of elongated compressed lobed and branched gray sporangia, arising in loose or compact clusters from branching strands of the membranous hypothallus, clothed with a thick, fragile, deciduous, universal covering of crystals of lime; 2 to 6 cm. long by 1 to 6 cm. wide and about 1 cm. thick; sporangium wall membranous, colorless or purplish, columella membranous, hollow, compressed, sometimes absent; capillitium a network of widely branching anastomosing stout purplish-brown or colorless threads, often with dark calyciform thickenings; hyaline at the extremities. Spores dull purple, strongly spinulose, 10 to 13 u in diameter (68).

2. *Physarum cinereum* (Batsch) Persoon.

Symptoms: Small gray or ashen-gray fruitbodies on grass blades, scattered in groups or rings over an area of several feet.

Distribution: Cosmopolitan, common.

Pathogen: Plasmodium colorless, watery-white, or yellow.

Sporangia sessile, closely gregarious or heaped, subglobose, elongate or plasmodiocarpous, more or less calcareous, lilaceous gray; wall simple, thin, more or less densely coated with lime; capillitium strongly developed of branching hyaline threads, the internodes more or less richly calcareous, the lime-knots rounded and angular. Spores clear violaceous, purplish-brown in mass, almost smooth to distinctly warted, 7 to 11 u in diameter (71, 68).

Epidemiology: Develop during humid weather in summer and autumn.

Control: Sweep and wash spore masses off with stream of water.

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